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OF

PRACTICAL MEDICINE

BY

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SIXTH EDITION

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PREFACE TO THE SIXTH EDITION.

In issuing the present edition I desire most gratefully to acknowledge the valuable assistance which I have received from my friends Dr. G. F. Crooke, and Mr. Malcolm Morris, in the preparation of the Sections on General Pathology and Diseases of the Skin, respectively. I trust, moreover, by the close and careful revision to which every page has been submitted, that the usefulness of the book has been increased, and rendered more worthy of the favour which has hitherto been accorded to it.

A. H. C.

33 Temple Row, Birmingham.
February, 1891.

PREFACE TO THE FIFTH EDITION.

Eight years ago, when issuing the first edition of this little work, I stated that my object had been partly to provide the student with a simple introduction to the study of systematic medicine; and partly to bring the essentials of the subject, as tersely and clearly as I could, within the grasp of those who for various reasons might not at the time be able to master a large standard treatise. No attempt has been made to compress a multiplicity of detail within a small compass, my intention has been to provide broad truthful sketches of the various types of disease, together with hints as to the principles of their management. Four large editions have successively become exhausted, and this —the fifth—has now been called for. Such generous favour is not only gratifying in itself, but entitles me to infer that the book in some measure meets a distinct need. The present issue has been revised to the best of my ability, certain sections have been entirely re-written, and some additions have been made.

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ELEMENTS OF PRACTICAL MEDICINE.

SECTION I.

GENERAL PATHOLOGY.

CONGESTION.

By *hyperæmia* or *congestion* is meant an overfulness of the blood-vessels, which may arise either from a *too abundant supply* of blood to a part, or some *interference with its proper escape.*

Congestion of the former type is called *active* or *arterial*, and occurs in one of two ways. The more common and important cause of active congestion is relaxation of the muscular walls, and consequent enlargement of the arteries of a part by disturbance of central or local vaso-motor nervous mechanisms. This is the explanation of the congestion produced by mechanical damage or irritation, the local application of heat, and blushing. Similar arterial dilatation follows the sudden removal of pressure, as in the rapid withdrawal of a large serous effusion, or in dry-cupping. A second and less frequent cause of active congestion arises from increased blood-pressure by obstruction of the circulation in adjacent arteries (*collateral congestion*). As a rule, congestion of this kind is soon righted by recovery of the tone of the distended vessels; hence, it is in the pulmonary vascular system, where the *tonus* is quite feeble, that the most striking examples of collateral congestion are met with.

Congestion arising from imperfect escape of blood is termed *mechanical* or *venous* when there is a direct impediment to the return of blood by the veins. It is a common result of uncompensated valvular disease of the heart, cirrhosis of the liver, direct pressure of a tumour upon important veins, and venous thrombosis. *Passive congestion* is closely allied to the foregoing, and is characterised by a sluggish and difficult circulation through the vessels of a part. It is met with as a consequence of weakness of the heart, leading to accumulation of blood in dependent parts under the influence of gravitation; or again, as a consequence of diseased conditions of the arterial walls obstructing the force of the heart's action.

The effects of *acute* and *mechanical* or *passive* congestion may be thus contrasted:—

Active Congestion.	Mechanical or Passive Congestion.
Redness and pulsation.	A dusky livid tint.
Elevation of temperature.	Temperature lowered.
Increased nutritive activity.	Diminished nutritive activity.
Functional excitement.	Functional depression.

Both varieties when long continued lead to swelling, serous transudation, and disturbed nutrition, with a liability to hæmorrhage.

HÆMORRHAGE.

Hæmorrhage denotes an escape of blood from the vessels; and may be occasioned by injury, disease, or faulty nutrition of the walls of the vessels, by alterations in the quality of the blood, by increased pressure of blood within them, or by one or more of these causes acting together. Special terms are applied to hæmorrhage from particular parts; thus *epistaxis* signifies bleeding from the nose; *hæmatemesis*—from the stomach; *hæmoptysis*—from the air-passages; *melæna*—from the bowels; *hæmaturia*—from the urinary organs; *metrorrhagia* or *menorrhagia*—from the uterus.

Hæmorrhage beneath a free surface forms more or less well-defined spots or small patches, which are called *petechiæ* or *ecchymoses;* when the effused blood infiltrates the substance of a tissue, it is called a *hæmorrhagic infarct;* and when it forms a tumour or swelling, it is called a *hæmatoma.* Those who are apt to bleed profusely from slight causes, are said to exhibit the *hæmorrhagic diathesis*, or to suffer from *hæmophilia.*

Hæmorrhage occurs in two separate ways. There may either be a distinct rupture of the vascular wall, in which case the bleeding whether large or small, takes place suddenly; or, on the other hand, as a result of loosening of the cement-substance between the cells of the capillary endothelium, the solid and liquid elements of the blood may gradually ooze out (*diapedesis*). The circumstances under which diapedesis occurs are not fully understood, but it probably always implies disturbance in the nutrition of the vessel-wall.

Blood which is extravasated and retained in the tissues, as a rule soon clots. The clot afterwards shrinks, the corpuscles disintegrate, and the liberated hæmoglobin undergoes chemical transformation, with changes of colour passing from brown to yellow. The residue may be absorbed, or a reactive growth of new tissue may take place, ultimately forming a pigmented mark or cicatrix, with perhaps a few crystals of hæmatoidin. When extravasation is attended with much damage to the surrounding tissues, suppuration is apt to occur, and the clot then breaks down into pus.

DROPSY.

Dropsy implies an undue accumulation of serous fluid in the serous cavities, or in the interstices of the tissues. Special terms are employed to indicate the extent and seat of the effusion. More or less generally distributed dropsy of the integuments is termed *anasarca;* when localised, *œdema; hydrothorax* denotes dropsy of the pleura; *hydropericardium*—of the pericardium; *ascites*—of the peritoneum; and *hydrocephalus*—of the cerebral ventricles.

The character of *dropsical fluid* closely resembles that of blood serum. It is colourless or straw-coloured; clear, or slightly turbid; alkaline or neutral; with a specific gravity of 1010—1014; with albumen, salts (especially chlorides), and extractives, in solution. Fluid of inflammatory origin is turbid, with more or less tendency to coagulate, of a specific gravity above 1014, a much higher proportion of proteids, and often contains flakes of lymph.

Among the causes of dropsy, mechanical venous congestion ranks first in importance. It gives rise to increased transudation of fluid by raising the pressure of blood in the capillaries, and interferes at the same time with the absorption of transuded fluid. Concurrent obstruction of the lymphatics greatly increases the effect of venous obstruction as a factor in dropsy. Thus, cardiac anasarca is no doubt partly due to obstructed outflow from the main lymphatic ducts, reflected back through the upper vena cava. Active arterial congestion, apart from inflammation, is not capable *per se* of producing dropsy. Defective nutrition of the capillary walls, by favouring the transudation of fluid in excess, is a second important cause of dropsy; and probably accounts in large measure for the dropsy of Bright's disease, chronic anæmia, and general debility. The operation of the foregoing causes is greatly enhanced by a weak circulation, undue laxity of tissue, want of nervous tone, and a poor and watery state of the blood.

Dropsy may be either local or general. *Local* dropsy most frequently depends upon mechanical obstruction of the veins leading from the dropsical part, as in ascites from cirrhosis of the liver, of phlegmasia dolens from plugging of the femoral vein. It occurs also as a result of inflammation, and (more rarely) of lymphatic obstructions. *General* dropsy may be either acute or chronic. Acute general dropsy begins abruptly, and results most commonly from acute Bright's disease, but it sometimes accompanies an acute general erythematous eruption on the skin, such as now and then occurs after a hearty meal of shell-fish, &c. Chronic general dropsy may be due either to (1) chronic disease of the heart or lungs, or (2) renal disease, or (3) anæmia and general debility.

When associated with weak or impeded circulation, general dropsy usually begins in the most dependent parts; otherwise, it begins in those parts where the tissues are naturally relaxed, as the under eyelid for instance. The integuments become swollen and pit on pressure. The vitality of dropsical parts is defective, and they are consequently liable to low forms of inflammation from slight causes. Dropsy of internal organs and serous cavities will be considered elsewhere.

DIAGNOSIS.—With regard to local dropsy it is always necessary to bear in mind, that when really local it must be due to a local cause. General dropsy at its onset is usually limited to a particular part, but then it develops *symmetrically* and affects corresponding parts on both sides.

The *acute form of general dropsy* may be distinguished by its abrupt onset, its frequent association with febrile symptoms, and its wide distribution. *Cardiac* or *pulmonary* general dropsy begins about the feet and ankles, gradually extends upwards, and is attended with the signs of disease of the heart or lungs. The skin is congested and dusky, and the superficial veins are more or less engorged. *Renal dropsy* begins in the loose connective tissue of the eyelids or scrotum, is widely distributed, and extends rapidly. The skin is usually pale, pasty, and anæmic, and the urine contains albumen and tube-casts. *Anæmic dropsy* is rarely extensive, and does not affect the serous cavities. It is usually confined to the ankles, but sometimes to the eyelids. Albumen is present in the urine only under very exceptional circumstances, and the general anæmic condition is obvious.

Treatment.—This should be first directed to the removal of the cause. Absorption of the fluid may be aided by increasing the secretions of the skin, bowels, and kidneys, by diaphoretics, watery purgatives, and diuretics respectively. Finally, the dropsical fluid may be removed by surgical interference—by means of tapping *(paracentesis)* in the cases of dropsy of the serous cavities; or by punctures or incisions, in anasarca.

INFLAMMATION.

The process of inflammation represents those changes which take place in a living tissue as the result of local injury; and therefore, any agent capable of inflicting damage may become a cause of inflammation. The phenomena of inflammation vary somewhat according to circumstances, but they may be conveniently considered under the following heads—(*a*) vascular disturbances, (*b*) exudation processes, and (*c*) changes in the inflamed tissues.

Vascular disturbances.—In the first place the arteries dilate and the blood-flow becomes accelerated. This dilatation extends to the capillaries and veins of the part, and soon afterwards the blood-flow becomes retarded; while the white blood-cells accumulate, and tend to adhere to the walls of the vessels. The current now becomes slower and slower, the vessels of the affected part become packed with corpuscles—both red and white—till at last complete stagnation occurs (*inflammatory stasis*).

Exudation processes.—Closely following the above disturbances, the white corpuscles—and the red also to a limited extent—migrate from the vessels by diapedesis into the surrounding tissues, accompanied with plasma which exudes at the same time. The fluid and the corpuscular elements which have escaped from the vessels, constitute the so-called *inflammatory exudation.* The nature of the exudation varies according to circumstances. When it speedily coagulates, it is said to be *fibrinous;* when in addition, there is more or less watery effusion it is termed *sero-fibrinous*, examples of which frequently occur in inflammation of serous membranes; while the watery or slimy exudation which accompanies certain inflammatory conditions of mucous membranes is termed *mucous.* The fate of the exudation varies. When it is small in bulk, and the surrounding tissue is little damaged, it is absorbed. When, however, the amount of exudation is too large to be properly nourished, and the surrounding damage is considerable, the inflammatory products degenerate, liquefy, and break down into *pus*, constituting the process of *suppuration.* Pus is an opaque yellowish fluid, of alkaline reaction, and average specific gravity of 1030. It consists of an albuminous fluid (*liquor puris*), holding in suspension innumerable cells, resembling for the most part white blood-cells, in various stages of fatty change. Micro-organisms usually abound, but their precise relation to the process of suppuration has not yet been established. Inflammatory exudation takes no active share in tissue-repair, but serves an important secondary purpose as a support for delicate granulations.

Tissue changes.—In the first place there are changes characterised by *necrosis* and *degeneration* which result partly from direct injury of the tissue elements, and partly from the stagnation of the circulation. Secondly, there are changes in the tissue immediately bordering on the inflamed area which are characterised by *increased nutritive activity*, and apparently represent efforts of nature towards the repair of damage inflicted. The former represent the direct results of damage; the latter, the results of irritation—each merging into the other by imperceptible degrees. Thus, the first effect of an irritant is to stimulate nutrition, and to promote tissue formation; but if it passes a certain point, the tissue-elements are damaged, and either degenerate or die outright. Again, an area of damaged tissue is always surrounded by a zone of irritated tissue, which is partly the distant and weaker effect of the primary injury, and partly the result of irritation set up by the damaged area itself. The changes usually described as the result of chronic inflammation—*e.g.*, interstitial pneumonia—are for the most part the result of irritation falling short of actual damage. The line which separates the two is obviously ill-defined and it is not surprising that the former in its later stages often passes on to the latter.

Where inflammation is slight and due to a temporary cause, it subsides without after effects. When slight and yet sufficiently persistent to cause slow and moderate damage, the latter is usually characterised by granular and fatty degeneration. When more intense, it is apt to cause suppuration, the tissue-elements and exudation breaking down by molecular necrosis into pus. If it be deep-seated an *abscess* is formed; but if on a free surface, it leads to *ulceration*. If the affected part be dry, and the inflammatory process slow, *caseation* may occur instead of suppuration. When very severe, inflammation causes death of tissue in mass, with formation of a slough (*gangrene*), leaving a deep ulcer on its separation.

Organisation and *repair* of inflammatory mischief begin in the nearest undamaged connective-tissue, by proliferation of its cell-elements, resulting in the formation of what is called *granulation-tissue*. The latter ultimately develops into fibrous tissue, or under certain circumstances into tissue other than fibrous.

VARIETIES.—Inflammation may be *acute* or *chronic*, according to its intensity, and rate of progress; *adhesive*, *suppurative*, *ulcerative*, or *gangrenous*, according to its effects; *circumscribed* or *diffuse*, according to its limits; *infective* or *simple*, according to its character.

The varieties of inflammation in mucous membranes may be conveniently described here. Three forms are generally recognised, representing different grades of intensity and depth to which the inflammation extends. The first and mildest form (*catarrhal inflammation*) is characterised by congestion and swelling of the membrane, desquamation of epithelium, and increased secretion. The latter at first consists of liquid exudation from the vessels, together with mucus, and is rendered more or less turbid from mixture with shed epithelium. If the process continues, an exudation of leucocytes takes place, which gives a semi-purulent character to the secretion (*purulent catarrh*); and if severe, erosions of the surface may occur (*catarrhal ulcers*). At any stage the inflammation may subside, or, on the other hand, it may become chronic. In the latter case, the membrane becomes thickened by fibrous hyperplasia, and the character of the surface becomes variously altered. In the second form (*croupous inflammation*) the process is more severe, the epithelium is partially destroyed, and the vessels are so damaged that a fibrinous exudation takes place on the surface, forming "a false membrane." The latter is readily removable, and has no tendency to become organised. In the third form (*diphtheritic inflammation*) the process is so severe that the epithelium is destroyed without desquamation, and becoming saturated with fibrinous exudation, undergoes a sort of coagulation, and forms a close-textured "false membrane." If moderately superficial, it is scarcely distinguishable from the croupous form; but if, as more often happens, the process is so severe as to also destroy the deeper layers of the membrane, the whole is involved in the "false membrane," which cannot then be removed, and when it separates by sloughing, leaves a well-marked ulcer. In course of time the latter undergoes cicatrisation, and may be covered with new epithelium.

SYMPTOMS.—*Locally*—redness, swelling, and heat. *Subjectively*—pain, tenderness, and disturbance of function. *Constitutionally*—the symptoms are those of fever, which, if suppuration occur, is usually of the hectic type.

THROMBOSIS.

THROMBOSIS denotes any coagulation of the blood in the heart or vessels, which takes place during life. The clot is called a *thrombus.* A thrombus, or ante-mortem clot, differs from that which is formed after death, in being paler, firmer, drier, and more granular in texture; by its greater relative proportion of white blood-cells and fibrin, and by its firmer adhesion to the parts with which it is in contact. When a thrombus forms slowly, it presents a laminated structure.

In all cases, except those in which the coagulation has been very rapid, as after ligature of a vessel, the thrombus first appears on the vascular lining membrane, slowly grows from without inwards by fresh accretions of fibrin, and finally blocks the vessel altogether. It now extends chiefly in a backward direction (*i.e.*, towards the heart) until it meets with a current of blood sufficiently strong to arrest its further progress.

Thrombi are proximately induced by one of two conditions, or by both combined, viz., *retardation of the blood-current* and *modification of the vascular walls.* The former may result from ligature, compression, or plugging of a vessel, aneurismal dilatations, solution in the continuity of a vessel from any cause, or cardiac feebleness; and the latter from endocarditis, arteritis, phlebitis, or atheroma. There are certain cases, however, of thrombosis, the etiology of which is by no means completely covered by the foregoing facts; such, for instance, as that which occurs in the femoral vein, towards the end of exhausting diseases like phthisis or cancer. It is probable that we have to do here with some antecedent nutritive disturbance of the blood, attended with breaking up of corpuscular elements, and consequent liberation of fibrin factors.

The thrombus may either become *organised* into connective tissue, by the extension of granulations from the wall of the vessel; or, if infective, it may *soften* (beginning in the centre) and break down into a yellowish puriform fluid, surrounded by the thickened coats of the vessel into which the suppurative process may extend.

Results.—1. The coats of the obstructed vessel become thickened and indurated, and if the thrombus become organised, the vessel is converted into a fibrous cord. 2. Certain circulatory disturbances occur:—thus, unless collateral circulation be quickly established, thrombosis of an artery will lead to the death of the parts supplied; while thrombosis of an important vein will cause swelling, and—if long continued—nutritive changes in the part which it drains. 3. A fragment of the clot may be washed off into the current of the blood, and lead to embolism.

EMBOLISM.

Embolism denotes the plugging of a vessel by a substance carried to it from a distance. The plug is called an *embolus*. The most frequent sources of emboli are detached fragments of thrombi in the veins; vegetations and morbid debris from the heart; drops of oil which have escaped from fat-cells after certain surgical injuries, etc., and have now become absorbed by the veins, and portions of new growths, or parasites.

An embolus, arising in one of these ways, is swept into the general current of the circulation, in which it flows on until its further progress is arrested by a vessel, the calibre of which is too small to allow it to pass. Thus, emboli which originate in the systemic veins, will be probably arrested in one of the smaller branches of the pulmonary artery; while those originating in the pulmonary veins or heart will be lodged in one of the systemic arteries; and those from the radicles of the portal vein, in the liver. It will thus be seen that, in all cases, the seat of the embolism is either in the arteries, or in the hepatic branches of the portal vein, or in their respective capillaries.

Results.—These vary according to the distribution of the occluded vessel, and the character of the embolus. When the embolus lodges in an artery which has free anastomoses, collateral circulation is quickly established, and no mischief results. When the obstructed artery is terminal—*i.e.*, with few or no anastomoses—as in the kidney, spleen, and brain, and (to some extent) in the lungs, the blood-supply is cut off from the area to which the vessel is distributed, and the part—which is conical in shape—necroses entirely or partially, constituting what is called an *infarct*. The infarct is surrounded by a zone of capillary congestion, in which punctiform hæmorrhages are apt to occur. It afterwards degenerates and is absorbed ultimately, leaving a depressed cicatrix. True infarcts of the lung are much rarer than usually supposed, and are not followed by necrosis. Hæmorrhagic infarctions (so-called) of the lung—according to Hamilton—are not of embolic origin at all, as hitherto taught, but small extravasations from rupture of alveolar capillaries, generally resulting from mechanical congestion, in connection with cardiac valvular disease. The wedged shape is due to the distribution of the bronchus and air-cells containing the extravasated blood. The latter is quickly re-absorbed, leaving no trace behind.

The above description applies to non-infective embolism. If, however, the embolus be infective, or in other words, if it be derived from a putrefactive source, as from a thrombus occurring in the neighbourhood of a pyæmic abscess, rapid softening and disintegration of the infarct takes place, resulting in the formation of an *embolic* or *metastatic abscess.*

HYPERTROPHY.

Hypertrophy denotes a quantitative overgrowth of an organ or a tissue. The increase in amount of the tissue may be due to an absolute increase in the *size of* the individual tissue-elements, when the hypertrophy is said to be *simple*, or to an increase in their number, when it is termed *numerical hypertrophy* or *hyperplasia*. The distinction, however, cannot be practically maintained, as they are usually associated together.

The anatomical changes in hypertrophy consist of increase in the size and weight, and sometimes in the shape of the organ affected.

Hypertrophy, in whatever way it may be induced, should be regarded as *the anatomical expression of increased functional activity*. Thus, in the case of muscular tissue, where hypertrophy is of frequent occurrence, any increase in the demands made upon the muscle by increased strain, or additional work, is responded to—*cæteris paribus*—by hypertrophy; and when it tends to bring about a balance between the work to be done and the means of doing it, it is regarded as *compensatory*. The hypertrophy of the bladder in chronic urethral stricture, and hypertrophy of the heart in valvular disease furnish examples of this. Persistent increase in the supply of blood to a part also leads to hypertrophy. The congestion induced by chronic irritation is undoubtedly an important factor in the tissue-overgrowth which occurs under these circumstances. Similarly, repeated irritation from intermittent pressure causes hypertrophy.

ATROPHY.

Atrophy implies a quantitative diminution of the substance of an organ or tissue, and is the outward visible sign of impaired functional activity. We speak of atrophy as being *simple* or *numerical*, according as the tissue-elements are merely reduced in size or diminished in number; but the two varieties are generally combined. It is impossible to draw a hard and fast line between atrophy on the one hand, and degeneration on the other, for the former so frequently leads on to the latter.

Apart from ordinary physiological atrophy, such as the shedding of hair and teeth, and senile atrophy, we may recognise three varieties:—(1) that which depends on defective nutrition; (2) that which depends upon disuse; and (3) that which depends on disturbances of innervation.

General atrophy is a good example of the first group, and is due to *insufficient supply of nourishment* as in starvation, constriction of some part of the alimentary canal, and defective digestion, assimilation, or absorption; or to *excessive waste*, as in fever, protracted diarrhœa, and immoderate hæmorrhage; or to some combination of these causes. In general atrophy the adipose tissue is first affected; then the muscles and viscera; and, lastly, the tissue of the nervous centres. Local atrophy from diminished blood-supply, and direct continuous pressure fall under the same head.

The best example of the second group is the atrophy which takes place in muscles from prolonged disuse. The disappearance of the ductus arteriosus and the Wolffian bodies after birth, and the shrinking of the mammæ after lactation furnish instances of physiological atrophy from disuse.

Atrophies from faulty innervation are classed as *tropho-neuroses*, on the assumption that nutrition is largely controlled by special nervous centres, from which trophic impulses are distributed along the nerves to the parts in connection with them. Any lesion of trophic nerve-centres or the nerve-fibres proceeding from them, leads to rapid atrophy and degeneration of the tissue under their control. The muscular wasting in infantile paralysis, from lead-poisoning, and from division or injury of peripheral nerves illustrates this form of atrophy.

DEGENERATIONS.

DEGENERATION differs from atrophy in that it is characterised by *qualitative* rather than by *quantitative* changes in the tissues. Two kinds of degeneration must be distinguished from each other. In the one, the albuminoid constituents of *tissues are chemically metamorphosed* into some new material of a lower type; while in the other, the *new material is deposited in the tissues* from the blood. The term *metamorphosis* is applied to the former, and *infiltration* to the latter variety. Both are attended with impairment of function, but to a much greater extent in the former than in the latter.

THE METAMORPHOSES.

These include cloudy swelling, fatty, lardaceous, mucoid, and colloid degenerations.

CLOUDY SWELLING is a form of degeneration to which the elements of various tissues and organs are prone, especially the muscles and the glandular epithelium of the liver and kidneys. It is a common result of early inflammation, and is also met with in febrile diseases in direct proportion to the intensity and duration of pyrexia. A typical instance of cloudy swelling is afforded by the condition of the renal epithelia shed in the early stages of tubal nephritis.

The affected cells enlarge and become granular, but clear up on the addition of acetic acid. Muscular fibres swell up, and lose their striæ. The nucleus is hidden by the granular deposit, but is not destroyed. Cloudy swelling is often associated with some fatty degeneration.

FATTY DEGENERATION.—This form of degeneration is characterised by the direct conversion of the albuminoid substance of cells and intercellular substance into fat. The fat appears in the form of minute oil-globules, of comparatively uniform size, and with little or no tendency to fuse together. The oil-globules are recognised by their sharp contour, dark colour, and solubility in ether. The cells or fibres at first enlarge, then break up, the albuminous matter between the oil-globules liquefies, and the fat becomes distributed in the tissues.

Fatty degeneration as a pathological process depends upon impaired nutrition, which leads to a retrograde decomposition of proteid matter. Such impairment may be occasioned either by direct interference with the vitality of a tissue, as in inflammation or phosphorus poisoning; or by depreciation in the quality of the blood, as in anæmia; or by a diminution in the blood-supply, as in obliterative arteritis and thrombosis.

CASEATION results from slow fatty degeneration, in the course of which a group of cells becomes converted into a yellow mass, of the consistence and appearance of cheese, and made up of minute fat globules and granular proteid matter. It is especially prone to affect cells crowded together in a confined space, as in syphilitic gummata, tubercles, and pneumonic exudations. Caseation always begins in the centre of such nodules—in other words, at that part which is most inaccessible to nutriment. A caseous mass, when once formed, acts like a foreign body, setting up reactive growth in the surrounding connective tissue, by which it is often completely encapsuled. In this state it may remain permanently quiescent, and ultimately become more or less calcareous. On the other hand, the caseous substance may soften and liquefy, forming an abscess.

LARDACEOUS OR ALBUMINOID DEGENERATION.*—In this form of degeneration, the tissue elements are converted into a peculiar homogeneous waxy-looking substance, called *lardacein*. The term amyloid is sometimes applied to this material, but it should be remembered that, unlike members of the starchy group, it is nitrogenous. In fact it is essentially a modified form of albumen, with some kind of affinity to fibrin. It exhibits a characteristic mahogany-brown colour when treated with a solution of iodine; while with a solution of methyl-violet it acquires a rosy-red colour.

Lardaceous degeneration occurs most commonly in the spleen, liver, kidneys, intestine, and lymphatic glands. From recent researches, the change appears to begin in the elements of connective tissue, more especially in the fibrous textures of small terminal arteries, and not—as has generally been taught—in the cells of glandular organs, into which it seems doubtful whether the change ever extends.

In the liver, the terminal branches of the hepatic artery are first affected, the walls of which lose their structure, swelling up and becoming homogeneous. The hepatic cells are compressed, and frequently atrophy and degenerate. In the kidney, the change begins in the vessels of the glomeruli. In the spleen and lymphatic glands, on the other hand; it generally begins in the lymphoid trabeculæ. But however the process begins, waxy-looking spots and patches are formed, the exact appearance of which differs according to circumstances. The affected organ or part gradually enlarges, and its consistency becomes much firmer. In an early stage the change can only be recognised with the microscope; but later on, it may be readily recognised by the naked eye, assisted by the use of reagents.

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* Some authorities say that the lardaceous process should be regarded as an infiltration.

The nature of the above degeneration, and the exact circumstances under which it occurs are not known. Clinically, however, it is almost always associated with grave cachexiæ, especially in connection with chronic tuberculosis, syphilis, long-standing suppuration, and dysentery.

Mucoid Degeneration.—Here the albuminous intercellular matrix of connective tissues becomes converted into a substance called *mucin*, giving to the affected part a mucilaginous or jelly-like character. Mucin differs from albumen in not containing sulphur, and being precipitated by acetic acid. It is met with most frequently in the inter-vertebral and costal cartilages of old people, and it often enters into the composition of connective tissue tumours, especially sarcoma.

Colloid Degeneration.—Here the albuminous contents of the cells (rather than the intercellular matrix) are converted into a jelly-like material, which differs from mucin in containing sulphur, and not being precipitated by acetic acid. This form of degeneration is especially liable to affect the cells of certain cancerous growths (*colloid cancer*); but it sometimes occurs in tumours other than those of a malignant kind.

THE INFILTRATIONS.

These include fatty, calcareous, and pigmentary infiltrations.

FATTY INFILTRATION.—In this condition, fat is deposited within the cells as distinct globules, which accumulate, increase in size, and tend to run together. The protoplasmic contents of the cells are at first merely *displaced;* but they subsequently atrophy, and are reduced to a small remnant, which is gathered around the nucleus. Typical examples of this condition are afforded by fatty infiltration of the liver, and pseudo-hypertrophic paralysis.

Etiologically, it results from an excess of fat in the blood, in greater quantity than can be got rid of by oxidation. This excess may arise, on the one hand, from a too liberal dietary, combined with insufficient exercise and fresh air; or, on the other hand, it may be due to the absorption of fat in excessive quantity, from rapid wasting of fatty tissues elsewhere, especially when associated with imperfect respiration, as in the fatty liver of phthisis.

CALCAREOUS INFILTRATION.—This consists in the deposit of calcareous particles, generally between but sometimes within the elements of the tissues. The particles are composed of lime and magnesian salts, and the process as a whole is closely analogous to the physiological process of ossification, with, however, certain distinctions, which may be thus tabulated.

OSSIFICATION.	CALCIFICATION.
Essentially an *active* process.	Essentially a *passive* process.
Increased nutritive activity.	Nutrition impaired.
Formation of new tissue.	Mere infiltration of old tissue.
Special structure (*lacunæ and bone corpuscles*).	No special structure.

Calcareous infiltration is apt to occur under circumstances of impaired nutrition. Thus, it is common in the walls of old phthisical cavities, in tissue damaged by slow inflammation, in the capsules of trichina spiralis, and as a result of senile malnutrition. The details of its causation are obscure.

Pigmentary Infiltration.—This is characterised by the deposit of pigmentary particles in the tissue elements. The black skin of the negro, and the choroid coat of the eye are physiological examples of this condition. Pathologically it occurs in the cells of certain sarcomas, in tumours growing from pigmented parts, and in the skin in Addison's disease. The pigment is called *melanin*, and is not derived directly from the blood, but is a metabolic product. An irregular form of pigmentation is met with in the lungs, which must not be confounded with pigmentary infiltration, viz., the black markings on section of the lungs of colliers, grinders, and others, resulting from the persistent inhalation of black particles in the form of dust. Arriving in the bronchi they penetrate the mucous membrane, and are carried into the lymphatic vessels and glands, where they remain permanently. Extravasated blood constitutes another source of accidental pigmentation.

FEVER OR PYREXIA.

Fever is a pathological condition characterised by undue elevation of temperature. The average temperature of the human body is 98·4° F. (36·8° C.), and only varies in health within very narrow limits. Of these variations the most important is that termed the "diurnal variation," which represents the interval between an early morning minimum, and an evening maximum. It rarely exceeds 1·5° F. Other slight variations also occur, due to the ingestion of food, external temperature, physical exercise, &c., but are clinically unimportant. The production of heat is exclusively traceable to the oxidation of tissues and food, which accompanies every form of bodily activity; while on the other hand, heat is being constantly lost by the lungs and excreta, and still more by conduction, radiation, and evaporation from the surface of the skin. The temperature of the body represents the balance between the production and loss of heat, both of which are under nervous control, and are also brought into constant relation with each other by the circulation of the blood. In fever this state of equilibrium is disturbed and the temperature rises. The increased heat of the body may be evident to the touch, but can only be correctly estimated by the thermometer. The increase of temperature varies from a slight rise, to 108° or even 110°. Below 101° the fever is slight, above 104° it is high, while over 106° it may be regarded as *hyperpyrexia*.

Associated with the elevated temperature, other phenomena are more or less constantly observed. The *skin* is generally hot, harsh, dry, and pungent; but it is sometimes bathed in profuse perspiration. Headache, vertigo, restlessness, insomnia, and delirium are common *nervous* symptoms. The *chill* or *rigor* which so frequently ushers in the febrile condition is also a nervous phenomenon, of a purely subjective kind, and is due to spasmodic contraction of the cutaneous vessels from shock. It is accordingly marked in direct proportion to the suddenness of onset of the febrile process. The *pulse* and *respirations* are usually quickened in proportion to the rise of temperature, while the characters of the pulse are variously modified. *Digestive disturbance* is indicated by a dry clammy mouth, a coated tongue, thirst, loss of appetite, nausea or vomiting, and constipation. The *urine* is scanty, high-coloured, of high specific gravity, and very acid; its solid constituents are both relatively and absolutely in excess; urea and uric acid are largely increased, while the chlorides are usually diminished. Lastly, *general emaciation* takes place in proportion to the intensity and duration of the fever.

TERMINATION.—The febrile process may terminate in death or recovery. In the event of recovery, the temperature subsides suddenly by *crisis*, or gradually by *lysis*.

VARIETIES OF TYPE.—1. Special terms are employed to denote varieties in the course and character of the fever; thus, it is said to be *continuous* when it persists for some time without marked variations; *remittent*, when between consecutive accessions of fever the temperature approaches but does not reach the normal level; *intermittent*, when between consecutive accessions of fever the temperature falls to the normal level; *relapsing*, when paroxysms of fever of considerable duration are separated from each other by periods of complete apyrexia lasting for one or more days.

2. Other varieties are based upon the severity and character of the febrile symptoms as a whole. Thus, it is said to be *simple*, when it occurs in a mild and trifling form; *inflammatory*, when it is dependent on acute local inflammations; *specific*, when it is supposed to be due to the introduction of a specific poison into the system.

Asthenic or *adynamic* fever is characterised by a comparatively slight rise of temperature, with symptoms of great weakness and prostration.

A fever is said to be *malignant* when it is exceedingly severe, and of a low and exceptionally fatal type.

The "*typhoid*" *state*, so-called because of its frequent occurrence in bad cases of typhus, is by no means confined to this affection. It denotes a low febrile condition which is especially characterised by a tendency to the accumulation of sordes about the lips and tongue; a dry brown tongue; great frequency, feebleness, and sometimes irregularity of the pulse; a liability to passive congestion of dependent parts; and low muttering delirium, ending in coma. It is supposed to be due to the defective elimination, and consequent accumulation in the blood, of the effete products of tissue-change.

Hectic fever mainly occurs in connection with persistent purulent discharge as that met with in caries of bones, phthisis, and the like. The fever is distinctly of an intermittent or remittent type, comes on insidiously, and is usually of long duration. It is further associated with marked progressive emaciation, circumscribed flushing of the cheek, a soft frequent pulse, profuse perspirations, and irregular action of the bowels. Hectic fever is now generally regarded as a form of septicæmia.

Nature of Fever.—From what has gone before, it follows that an elevation of temperature might arise either from diminished heat-loss, or from increased heat-production, or from disturbance of the heat-regulating nervous mechanism. According to the recent researches of Macalister, the whole fabric of the thermo-nervous mechanism is liable to disturbance in fever, though to a variable extent under different circumstances. The heat-regulating centres are the first to suffer, and are always implicated; then there is always a relative and sometimes an absolute increase in the amount of heat produced, also the result of disturbed innervation; and lastly, the centres controlling heat-discharge may become affected, and if the heat-loss be seriously interfered with, hyperpyrexia is apt to supervene. The systemic disturbances met with in fever are to be regarded as entirely of secondary origin.

RELATIONS OF MICRO-ORGANISMS TO DISEASE.

In many infective diseases—though not as yet in all—the existence of minute living organisms has been established beyond all doubt. The question arises as to what is the precise nature of the connection of such organisms, if any, with the diseases in which they are found. The so-called germ theory assumes that all infective diseases are directly due to the presence and development within the body of specific living contagia or germs. This theory is mainly based on the capacity of such contagia for indefinite multiplication within the body, however small the quantity which is originally introduced; on experimental and clinical evidence that the contagium of any one disease produces that disease and no other; on the latent period which separates the moment of exposure to the contagious influence from the actual appearance of symptoms; on the definite course of such diseases; and lastly, on the experimental causation of at least one infective disease (anthrax) by the inoculation after repeated artificial cultivation outside the body, of living organisms originally derived from a similar case. Admitting this view, every such organism is distinguished from an ordinary animal or vegetable parasite by its capacity to modify—in a manner specific to itself—the various physiological processes of the body.

The micro-organisms now under discussion go by the generic term of *bacteria*. They are of a vegetable nature, belonging to the order of Fungi, and are of various kinds. Those known as *micrococci* consist of exceedingly minute spherules isolated from each other, or arranged in rows or in masses (*zooglœa*); others resemble minute straight rods, also existing separately, or in rows, or in masses, and are called *bacilli;* while in a third variety the rods exhibit a spiral shape, and are termed *spirilla*. They multiply either by transverse subdivision, or by the formation of minute bright-looking particles called "spores," which under favourable circumstances, develop into bacteria similar to those from which they were derived. The growth and development of bacteria demand a due supply of nutritive material, of which part at least must be of an organic nature; a certain degree of moisture; and moderate warmth. Bacteria are destroyed by the temperature of boiling water; though it is probable that the spores in many cases are able to resist a higher temperature with impunity. Many substances like corrosive sublimate, carbolic acid, &c., exert a similar destructive effect. Their mode of action on the media in which they live and grow is probably closely allied with that of fermentation.

Vegetable organisms answering to this description are found in abundance almost everywhere—in the soil, in the water, and in the air. It is clear, therefore, that enormous quantities must be introduced into the body with the food we eat, the water we drink, and with the air we breathe. Of the bacteria thus introduced the vast majority are either incapable of development, or are altogether destroyed by reason of the unsuitability of their new surroundings. But some are able to multiply freely in the healthy body; and others while incapable of this, may develop when the tissues are morbidly changed in such a way as to conform with their requirements, constituting a *predisposition* on the part of the system.

It is obvious that the latter group alone has any pathological significance. Assuming that a bacterial settlement of this kind has actually taken place, prejudicial results quickly follow. For instance, the organisms draw on the tissues for the materials necessary to their growth; the secondary products of their vital activity are introduced into the system; the normal vito-chemical changes of the tissue elements become disturbed; and the organisms themselves by getting into the current of the circulation may give rise to embolic infarction. Sooner or later the process ends either by reason of an amount of functional disturbance incompatible with the life of the patient; or by the rallying of the normal powers of the tissues, and the establishment of conditions which are no longer consistent with the development of the bacterial organisms. It is a curious fact that diseases of this kind leave the system in a condition which is peculiarly insusceptible to a repetition of the same bacterial growth, and thus a more or less complete immunity from the recurrence of the disease in question is conferred upon the patient.

COLLAPSE AND SYNCOPE.

Both collapse and syncope are characterised by functional depression, in the former—of the nervous system, in the latter—of the heart, and they are met with under a great variety of circumstances. Either may arise from the operation of some specific poison in the system or as a result of high fever, suppression of certain excretions, violent mental motion, excessive pain, rupture or perforation of internal organs, hæmorrhage and the like.

The *symptoms of collapse* are as follows:—the temperature usually (not always) falls; the skin is pale, cold and clammy, the features are pinched and drawn; the respiration is slow, shallow and often sighing; the pulse is small and feeble, sometimes very rapid, sometimes very slow; hiccup, nausea or vomiting, noises in the ears, dimness of sight, and extreme debility, are also common symptoms. The mental condition varies; it may be clear, or insensibility may be partial or complete.

Syncope differs from collapse mainly in the suddenness of its onset, the greater intensity of its symptoms, and its shorter duration. There is no time for a marked depression in the temperature.

DEATH.

The circumstances under which death takes place are very complex and variable. Certain typical modes of death may be distinguished with advantage, though it is not always possible to refer any given death exclusively to one mode. Death may begin at the heart, lungs or brain.

When the heart's action suddenly and completely fails under any of the circumstances enumerated above, death is said to take place from *syncope*. When death is preceded by marked general depression and prostration, it is said to occur from *collapse*. When the action of the heart ceases more gradually, together with a similarly gradual failure in the general vitality, death is said to occur from *asthenia*. When death is due to interference with the respiratory functions, it is said to occur from *asphyxia*. Lastly, death from *coma* begins at the brain, and is characterised by stupor and insensibility.

SECTION II.

GENERAL DISEASES. (A).

TYPHUS FEVER.

TYPHUS FEVER is undoubtedly produced by a specific poison. As regards its origin, Murchison and others are strongly of opinion that it may arise *de novo* from overcrowding. Whether this be so or not, typhus fever is eminently infectious, and is easily communicable by the poisons contained in the emanations from the patient. The contagious influence, *per se*, travels but a short distance, being readily lost by dilution with the air; but it may be conveyed a considerable distance by contaminated clothing, furniture, &c. A feeble state of health, defective hygienic surroundings, bad ventilation, want of cleanliness, impure water and air, and intemperate habits, appear to favour the development of the disease. Typhus occurs most frequently in those of middle and advanced ages.

MORBID ANATOMY.—The *post-mortem* appearances are characterised by *disorganisation*. The blood is dark and coagulates imperfectly; the muscles are softened and degenerated; and many of the internal organs become enlarged, hyperæmic, and softened.

SYMPTOMS.—The period of *incubation* is said to be about nine days, and may be marked by premonitory signs of constitutional disturbance.

Typhus varies much in severity. In a typical case, the *invasion* is generally abrupt, and is ushered in with chilliness, or rigors, followed by high fever. Prostration and depression are present from the outset, in direct proportion to the severity of the case; and the general character of the disease is that of acute blood-poisoning of a low type. Nervous symptoms are prominent and come on early. Almost from the first there is much restlessness with sleepless nights. The head is heavy and confused, and complaint is also made of giddiness, intolerance of light, and singing in the ears. Delirium is also an early symptom, and though at first it amounts to mere mental dulness and confusion, it soon becomes marked, and is usually of a low muttering type. The expression is dull, and the complexion dusky. The limbs are often painful, and tremble on movement. The tongue trembles when protruded; it is covered with a white or yellowish-brown fur, and rapidly becomes dry. There is often an unpleasant taste in the mouth. Thirst, nausea (occasionally vomiting), with almost complete loss of appetite, are frequent symptoms. The bowels are generally somewhat confined; but when relaxed, the stools are dark coloured, and quite unlike the pale shreddy evacuations in typhoid fever. The pulse is quick, soft and compressible. The urine is diminished in amount, and high-coloured. Symptoms of catarrh, such as sneezing, slight cough, and expectoration, are not uncommon. The temperature will be considered separately.

The eruptive stage.—The eruption consists of *maculæ* or "mulberry-spots," usually combined with more or less *sub-cuticular mottling*. The *maculæ* appear about the fourth or fifth day on the front of the chest and upper part of the abdomen, and if abundant, on the limbs also. They come out in one crop, and persist throughout the disease, and also after death in fatal cases. They consist of irregular round reddish spots, which (when fully developed) are hæmorrhagic, of a dark-red mulberry-juice colour, and do not disappear on pressure. The *mottling* is of a dusky-red colour, and appears as if beneath the surface of the skin.

In this stage, the previously enumerated symptoms tend to increase in severity. The depression becomes still more complete. The delirium is apt to lapse into semi-coma, or stupor, and the special senses are dulled. The tongue becomes dry, brown, and cracked; and the lips and teeth are encrusted with sordes. Thirst persists, and the appetite is completely lost. The pulse rises to 130 or 140, or more, and is very feeble, fluttering, and dicrotous, the first sound of the heart becomes indistinct or even inaudible—indicative of threatening cardiac failure. Respiration is hurried and disturbed. The urine is very scanty, high-coloured, and not infrequently contains albumen. In bad cases it is often retained, or dribbles away involuntarily with the stools. Bed-sores are common.

Stage of decline.—The average duration of typhus fever is from 14 to 21 days. Death is most frequent about the end of the second week, either as the result of profound exhaustion, or of some complication, such as pneumonia. In favourable cases—which are the rule—the fever ends by *crisis*, in the third week. The patient falls into a good sleep, from which he awakes greatly relieved. The tongue moistens, the mind clears, the appetite returns, and recovery often sets in with wonderful rapidity.

Course of the temperature.—There is a rapid rise of temperature from the first, reaching a maximum of 104° or 105° by the fourth or fifth day, with only slight morning remissions. About the sixth day a remission takes place in all but the most severe cases. Then a second rise—but not up to the first maximum—which is maintained in favourable cases till the final fall, which is sudden and complete. In fatal cases, however, death is often preceded by a rise to 108° or 109°.

The *complications* of typhus are due, for the most part, to the prostration, disorganisation, and toxæmia which characterise this fever. Thus cardiac softening and degeneration lead to extreme feebleness of the circulation, which in turn creates a liability to hypostatic congestion, most frequently affecting the lungs, and passing on to pneumonia. More rarely, local infective mischief may give rise to parotid bubo, abscesses, gangrene, or renal disease.

The main *diagnostic* features are—the rapid rise of temperature with imperfect morning remissions; the early prostration; the early appearance of nervous symptoms of a low type; and the "mulberry" rash.

As to *prognosis*—advanced age, previous intemperance, great feebleness or extreme rapidity of the pulse, profound coma, hiccup, suppression of the urine, delirium of a marked kind alternating with stupor, are very unfavourable. Of the complications, pneumonia and parotid bubo are the most ominous. A large majority of all cases recover.

TREATMENT.—The patient should rest completely in bed, in a well-ventilated airy room. The food should be liquid, nutritious, and easily assimilable. Alcoholic stimulants are needful in most cases; but are especially called for in the aged, and in those whose vitality is impaired by excesses of any kind. The secretions must be attended to, and the urine should be drawn if necessary. Medicinally, the mineral acids, quinine, and tincture of steel, have been chiefly recommended. The *nervous symptoms* are best met by tepid spongings of the body, and the application of ice to the head; *violent delirium with excitement*—by tartar emetic and opium; *low muttering delirium* and the "*typhoid*" *state*—by stimulants (opium is contra-indicated); *stupor*—by stimulants, or strong coffee, and by counter-irritation to the nape of the neck.

TYPHOID FEVER—ENTERIC FEVER.

Typhoid fever is due to a specific poison, and is not communicable by the exhalations from the patient. Eberth and others claim to have discovered specific bacilli in the mesenteric glands, spleen, &c., but their precise relation to the disease has yet to be made out. The source of the poison is to be found in the emanations from decomposing typhoid stools, and also—according to Murchison—from decomposing sewage matter of any kind. It is mainly conveyed through the medium of drinking-water, which has been contaminated by soakage from privies, cesspools, or drains. The disease is most common between the ages of 15 and 25 years.

Morbid Anatomy.—The most characteristic lesions are those affecting the lymphatic structures of the abdomen:—1. *Inflammation and ulceration of the solitary and agminated glands (Peyer's patches)* of the intestine. These changes begin and are most extensive in the neighbourhood of the ileo-cæcal valve, becoming less marked on proceeding upwards from this point. The solitary glands of the large intestine are frequently affected. The glands first enlarge from proliferation of their cellular elements. During the second week the inflammation may subside, and much more frequently the infiltrated tissue undergoes necrosis and forms a slough, which becomes detached, either entire or in fragments, in the course of the third week leaving an ulcer. The typhoid ulcer may be simultaneously described and contrasted with the tubercular intestinal ulcer, thus—

The *typhoid ulcer* has its long axis parallel to the intestines; its edges are thin and undermined; it never leads to stricture of the bowels; it generally heals, and is accompanied with little or no peritonitis.	The *tubercular ulcer* has its long axis transverse to the intestine; its edges are thick and indurated; it is often followed by stricture of the bowel; it rarely heals; and is always accompanied with local or general peritonitis.

The floor of the ulcer is formed by the sub-mucous, muscular, or peritoneal coats of the intestine according to its depth. Cicatrisation takes place, as a rule, during the fourth week; but the glands and mucous membrane which have been destroyed, are not renewed. *Perforation* of the intestine may occur either from gradual extension of the ulcer, separation of the whole thickness of the gut by sloughing, or from laceration. *Hæmorrhage* from the bowel is also not an uncommon event. Early hæmorrhage (occurring during the first 10 or 12 days) is generally slight, and depends upon vascular rupture from extreme congestion of the intestinal mucous lining. Late hæmorrhage (most frequent during the third week) is usually abundant, and results from erosion of larger vessels in the floor of the ulcer. 2. The *mesenteric glands* become infiltrated, enlarged, and softened. 3. A similar *enlargement of the spleen* is very constant.

As in all continued fevers, the muscles and glands of the body are apt to undergo more or less granular degeneration.

Symptoms.—The period of *incubation* extends from 10 to 20 days, but it is not attended with any marked symptoms.

The *actual attack* begins insidiously. The patient is feverish, and at first complains of little more than frontal headache, abdominal discomfort, general malaise, and aching pains in the limbs. Slight epistaxis is also not uncommon.

At first the symptoms are not, as a rule, so severe, as to compel the patient to take to his bed; but the general condition soon becomes more characteristic. The countenance is pale, though the cheeks may be flushed; the skin is hot and dry; the frequency of the pulse is generally much less than during the invasion of other fevers—often under 100, when the temperature is above 104°; the appetite is lost; and thirst is marked. The tongue is usually coated with a coarse white fur, which thins off towards the tip and edges, and is often cracked transversely. The following abdominal symptoms may now be usually observed:—*Pain* and *tenderness*, especially over the right iliac fossa; *tympanitis*, with gurgling on pressure over the right iliac fossa; *enlargement of the spleen;* and *diarrhœa*. The behaviour of the bowels is not constant. Usually they are relaxed from the outset, the stools—which are at first fæcal—gradually assuming the appearance described below. But, not infrequently the bowels are confined in the first instance, becoming relaxed later on; while, in a third group (especially common in children), they are confined throughout.

Typical typhoid stools are of thin consistence and pale yellowish colour, resembling pea-soup, of an offensive odour, and of alkaline reaction. On standing, a deposit occurs, which consists of epithelial debris, blood, shreds of mucous membrane, undigested food, and crystals of triple phosphates; while the supernatant liquor contains albumen, and salts (especially chloride of sodium) in solution. In this stage nervous symptoms are not prominent.

The *eruption* begins to appear from the seventh to the tenth day, upon the abdomen chest and back; it comes out in successive crops, each spot lasting for three or four days. The number of spots varies much. The average duration of the rash as a whole is about a fortnight. The spots are roundish, of the size of a millet seed, slightly elevated, well defined, rose coloured, and disappear completely on pressure, and after death.

During the *second week* the countenance becomes more flushed, and the patient rapidly emaciates. The frontal headache disappears, but he gets more apathetic and restless, and lies mostly on his back. All muscular movements are feeble and uncertain. There is often sleepiness with some delirium, but at this stage the latter rarely gets beyond slight rambling. The tongue becomes dry and cracked in the centre, and trembles when protruded; the abdominal symptoms increase, and hæmorrhage from the bowels may occur. The pulse becomes more frequent, and feeble. There is usually some little cough and expectoration; and the physical signs of bronchial catarrh may always be detected, especially over the posterior bases of the lungs.

In the *third week*, the patient is at his worst. Dull, worn, and prostrate, he lies hopelessly on his back; the tongue is brown and dry, and often covered with sordes which extend to the teeth and lips; the pulse is very rapid, feeble, and generally dicrotous; the delirium becomes more marked; and in bad cases there may be marked stupor. The fæces and urine are often passed involuntarily, or the urine may be retained. It is about this time that severe hæmorrhage and perforation are most apt to occur.

In course of the *fourth week* the symptoms usually subside in favourable cases. Defervescence is by *lysis*. The total duration varies considerably, in very mild cases it may be less than four weeks, while in severe cases it may be much longer. The convalescence is slow, and liable to interruption from relapse.

In relapse, all the phenomena above described reappear, usually (but not always) in a milder form, and of shorter duration than the primary attack. There is no constancy as to the duration of the interval; which, in my own cases has varied from 2 to 18 days. Sometimes there is a second or third relapse. In a case of mine, in which 18 days of pyrexia separated the primary attack from the first relapse, there were three relapses.

Temperature.—The primary onset of the fever is very characteristic; the temperature rises about 2° every evening, and falls about 1° every morning for the first four or five days, attaining a maximum towards the end of the first week. During the second week, this maximum is usually maintained, and the fever is of a continued type; during the third week it is of a remittent type; while during the fourth week it is of an intermittent type. The more severe the case, the longer the duration of the period of continued fever. During convalescence the temperature occasionally exhibits a sudden temporary rise, which must not be confounded with "relapse."

The most important *complications* are general bronchitis, pneumonia, pleurisy, perforation of the bowel, intestinal hæmorrhage, and peritonitis; while phlegmasia dolens, prolonged mental impairment, laryngeal ulceration, or grave anæmia may occur as *sequelæ.*

The *diagnosis* is often very difficult, especially during the first few days. A rule of considerable service in practice, is that any acute fever, lasting for a week, without any rash or apparent local inflammatory mischief, is probably either typhoid or tuberculosis. Typhlitis and ulcerative endocarditis are also liable sometimes to be confounded with typhoid. It should be remembered that typhoid is sometimes much milder and of shorter duration than the type above described, especially in children.

The *prognosis* is favourable on the whole, but the possibility of death from perforation or hæmorrhage gives an element of uncertainty to every case. Protracted high temperature after the end of the second week, and steadily progressive frequency of the pulse, with marked dicrotism, are of grave import.

Treatment.—The *hygienic* management is similar to that of all fevers. With regard to the *diet*, great caution should be exercised. The food should be given exclusively in a liquid form. A milk diet is the best, to which may be added beef-tea, arrow-root, custard, or jelly. Beef-tea should, however, be avoided if the diarrhœa be excessive. A caution is also necessary against the indiscriminate use of milk in large quantities; hard curds are often formed in the stomach, which on passing into the intestine may cause symptoms of violent irritation, and do much harm. Cooling drinks may be given in moderation. I have often found a tablespoonful of black coffee, from time to time, relieve the thirst better than anything else, and it has the advantage of not interfering with digestion. *Alcoholic stimulants* are not needed in ordinary cases, and few require them before the end of the second week. A rapid fluttering pulse and inability to take proper nourishment, provide the safest indications for their use. Again, during convalescence, a glass of light wine with the heavier meals often aids digestion and accelerates recovery. There is no doubt that many cases will recover without medicinal treatment, but from careful observation of a large number of cases and many methods of treatment, I have formed a very high opinion of the value of turpentine in doses of ♏x.-xv. every four hours. Some still prefer the mineral acids to other drugs. During the second week—and after—when the temperature runs high, antipyretic treatment by large doses of quinine, or by cold baths, has been strongly recommended. The *diarrhœa* should not be checked unless it be excessive, when five to ten grains of Dover's powder, or an opiate enema may be given occasionally. If the bowels be *constipated*, great care is requisite in the use of aperients. Nothing beyond a small dose of castor oil (which can be repeated if necessary), or a simple enema should be employed. *Hæmorrhage* may be best controlled by the application of ice to the iliac region, by cold enemata, and by the internal administration of turpentine, acetate of lead with opium, gallic or sulphuric acid, hazelin or ergotin. During *convalescence*, food should be given in small quantities at a time, and at moderate intervals, indigestible substances being strictly avoided. No solid food should be given until at least one week after the temperature has become normal.

RELAPSING FEVER.

This is a peculiar specific fever, which is characterised by periodical intermissions and relapses. In whatever way the disease may first arise, it is exceedingly infectious, and as a rule it takes the form of an epidemic. It especially occurs during seasons of famine, and its propagation is enhanced by overcrowding and insanitation. Its incubation is probably under nine days.

Minute corkscrew-shaped organisms (*spirilla*) have been found in the blood during the febrile periods, and are regarded by some as the specific cause of the disease.

Symptoms.—The *invasion* is remarkably sudden, setting in with rigors, frontal headache, marked pains in the limbs, and general depression. Febrile reaction soon follows, with its usual accompaniments. The more characteristic symptoms at this stage are—severe nausea and vomiting of a bitter bilious fluid, with pain at the epigastrium. The tongue is furred, and may become dry and brown. The bowels are confined, and there is usually more or less jaundice. The pulse is very frequent, and in bad cases feeble and irregular. The urine is febrile. *There is no eruption.*

The general condition now appears grave indeed; but about the fifth or seventh day, a profuse perspiration breaks out, the pulse falls to its normal standard, the fever disappears by *crisis,* the tongue gets moist, and the appetite quickly returns. The patient fancies himself well, and looks forward to a speedy and complete recovery—but in this he is disappointed. Generally in about seven days (fourteen from the first onset) there is a *sudden relapse,* with the reappearance of all the former symptoms. The intensity of the relapse is, generally less than that of the first attack. The relapse continues from three to five days, when a *second crisis* occurs. This may be repeated even a third or fourth time. The debility which results is considerable, and the convalescence is slow.

The *temperature* rises rapidly to a maximum of about 105°, which is reached on the fourth or fifth day, and is maintained with slight morning remissions until the critical fall. During the relapse, the temperature does not usually rise so high as before.

Troublesome *complications* are apt to occur, of which bronchitis, severe pains in the limbs, ophthalmia, anasarca, and diarrhœa are the most important. Pregnant women always abort. The *prognosis* is usually favourable.

TREATMENT.—No specific form of treatment is known. At the outset, gentle aperients, with cooling drinks, a light nutritious diet, and perfect rest are required. Tepid sponging often gives great relief. Opium is said to greatly relieve the headache, muscular pains and insomnia. It may be advantageously combined with belladonna, or given hypodermically. Quinine has no influence in preventing the relapse.

SCARLET FEVER

Scarlet fever is produced by a specific poison. It is extremely infectious. The poison is contained in the emanations from the patient, and in the epithelial scales which are so freely shed towards the close of the disease. No limit is known to the period for which contaminated clothes, &c., may retain their infective properties. A second attack is rare. The disease is much more frequent among children than adults. There are no characteristic anatomical appearances.

Symptoms.—The period of *incubation* is exceptionally short, and may not exceed twenty-four hours. The *invasion* is very sudden, and ushered in by chilliness, vomiting, or (in infants) convulsions. The temperature rises rapidly to a high grade, and is accompanied with the usual febrile symptoms. The pulse is *extremely rapid.* The tongue is furred on the dorsum, but the tip and edges are bright red. The throat is sore, red and relaxed. The redness is *uniformly diffused* over the fauces, soft palate, and back of the pharynx, which distinguishes it from the one-sided inflammation of tonsillitis. Slight nocturnal delirium is not unusual.

The *eruption* first appears about the neck and shoulders on the second day, and extends thence over the whole body in the course of twenty-four hours. It consists at first of bright red unelevated spots, which soon fuse together, till the skin becomes of a bright scarlet hue, readily dispelled by pressure. The rash reaches its height in three or four days, and soon after begins to fade. It disappears altogether by the beginning of the second week; and in mild cases, still earlier.

The soreness of the throat increases, and a thin exudation over the fauces, with some slight ulceration, is often seen. The glands at the angle of the jaw are always more or less enlarged and tender, and the surrounding parts are often œdematous. The temperature attains its maximum with the rash, and falls to normal as the latter disappears. The pulse is similarly affected. During the eruptive period, the tongue cleans from before backwards, and becomes of a bright red colour, with large prominent papillæ (*strawberry tongue*). The bowels are usually constipated; the urine is of febrile type, and may contain a little albumen. In simple cases, nervous disturbance is limited, as a rule, to headache and some nocturnal delirium.

Desquamation, or peeling of the skin, begins soon after the rash has faded. Where the skin is thin, the scales are small and branny; where it is thick, as on the hands and feet, it comes away in large flakes. Its duration varies greatly according to the treatment adopted, but it usually extends over two or three weeks. The symptoms now completely subside; the urine becomes more abundant, and may contain a quantity of renal and vesical epithelium. The temperature is often subnormal during the earlier part of this stage.

VARIETIES.—1. *Simple* scarlet fever, as already described. 2. *Scarlatina anginosa.* Here the throat symptoms are especially severe; active ulceration takes place, and may extend to the adjacent parts of the naso-pharynx, and to the nasal cavities; the glands at the angle of the jaw greatly enlarge; and abscesses are apt to form in the neck and elsewhere. 3. *Scarlatina maligna* is characterised by grave nervous symptoms of a low type from the first, viz., great prostration, delirium, with convulsions or coma. The pulse is rapid, feeble, and irregular. The rash is dusky and comes out badly. This variety is extremely fatal.

Complications are of frequent occurrence. Of these, the most important is *acute nephritis*, which is apt to supervene during the second or third week from the beginning of the fever. It is characterised by albuminuria and dropsy; and very often the urine is dark-coloured or smoky from the presence of blood. Recovery takes place as a rule, but a fatal result is not uncommon, and in some cases albuminuria may persist for weeks or months after all other symptoms have disappeared. *Purulent inflammation of the middle ear*, extending from the throat along the Eustachian tube is also a common occurrence. Amongst other complications the following may be mentioned:—abscesses in the neck, rheumatic inflammation of the joints, pleurisy, and pneumonia. The inflammations of the pleura and of the joint-cavities are probably of pyæmic origin, and the effusions which result are very apt to be purulent.

The *diagnosis* of scarlet fever from other eruptive fevers will be given further on. The fact should not be forgotten that erythematous eruptions sometimes complicate the early stages of certain acute diseases—such as small-pox for instance—and considerable care is often necessary to interpret them aright.

The *prognosis* of simple uncomplicated scarlet fever is generally favourable; and the gravity of any particular case is almost always in direct proportion to the number and importance of the complications.

Treatment.—The patient should be isolated in a well-ventilated room, from which all superfluous furniture should be at once removed. At the outset, to reduce the fever and to ease the throat, nothing is more useful than chlorate of potash combined with tincture of aconite, which should be given frequently. Arsenic has also been highly recommended. Topical applications may be used with advantage for ulceration of the throat and nose. For this purpose, solutions of borax, carbolic acid, chlorate of potash, and Condy's fluid are the best. Great relief to the throat is often obtained by allowing small lumps of ice to dissolve in the mouth, and by the external application of hot fomentations. Tepid or cold water spongings are especially useful to allay nervous symptoms. During desquamation, daily bathing, followed by inunction of the whole surface with carbolic oil—two per cent.—should be employed. In the malignant variety, nourishing food stimulants and quinine are specially needed. Complications require treatment as they arise.

MEASLES—MORBILLI.

Measles is an acute specific disease, dependent upon a specific poison which is contained in the emanations from the patient. It may also be communicated by clothing, &c., but the contagium of measles is much less persistent than that of scarlet fever. A second attack is rare. Children are especially apt to be affected. There are no characteristic anatomical changes.

Symptoms.—The period of *incubation* lasts usually from 7 to 10 days. The *invasion* sets in with chilliness, or (in infants) with convulsions, but not so suddenly as scarlet fever. The fever rises more or less rapidly to 101° or 102°, or higher in grave cases; but it very rarely equals that of scarlet fever. At the same time, the symptoms of ordinary catarrh of the nose and eyes are observed, indicated by redness, clear watery discharges, and frontal headache. Some intolerance of light, and epistaxis are not unusual. The catarrh soon increases, and extends into the throat, air-passages, and alimentary canal, causing sore-throat, hoarseness, cough, expectoration, slight difficulty of breathing, and wheezing sounds in the chest; loss of appetite, gastric discomfort, and constipation; but occasionally the bowels are relaxed.

The *eruption* appears on the fourth day, first on the forehead, thence spreading to the rest of the body. It consists of little irregular patches of a dusky-red colour, and slightly elevated. At first the rash disappears completely on pressure; but when it is very intense, or has been out for some time, a yellowish staining may persist after pressure. It lasts altogether four or five days, and is followed by an insignificant powdery desquamation.

The catarrhal symptoms persist, but the character of the discharges from the mucous membrane now alter. They are no longer clear and watery, but turbid, thick, yellowish. The margins of the eyelids are red and tumid, the tongue is usually moist, thickly coated, and often "patchy" in appearance. There may also be some vomiting and diarrhœa at this stage. The temperature often remits a little on the appearance of the rash, then rises to a maximum with the rash, and usually ends by crisis about the seventh or eighth day. The urine is febrile.

VARIETIES.—1. *Simple measles* as above described. 2. *Malignant measles*, which exhibits analogous symptoms to those enumerated with malignant scarlet fever. In this form the eruption is imperfect, and is very dark and dusky. Hæmorrhages are also apt to occur from the cutaneous and mucous surfaces.

The most important *complications* are:—capillary bronchitis, catarrhal pneumonia, pulmonary collapse, chronic inflammations of the eye, ear, or nose, and gangrene of the mouth or vulva.

The *diagnosis* will be afterwards considered. The *prognosis* is nearly always favourable in this country, unless it be complicated, or of a malignant type.

No *treatment* of a special kind is necessary in simple cases, beyond the relief of symptoms. Any threatening of the "typhoid state" should be met at once by the free use of stimulants, and nourishing food. If there be any hereditary predisposition to tubercle or struma, measles is very apt to become the starting point of actual tubercular or strumous disease. Accordingly, it is very desirable that the patient should be placed during convalescence under tonic and invigorating conditions of life.

EPIDEMIC ROSEOLA.

Epidemic Roseola, or German measles, is an infectious eruptive fever, of more common occurrence among children than adults. It is almost always a trifling affection, and its chief claim to notice here, lies in the possibility of its being mistaken for measles. Nothing is known precisely of its pathology.

Slight febrile symptoms usually precede the appearance of the eruption for about one day. The rash first appears on the nose and cheeks in the form of small red spots, varying in size, round or oval in shape, slightly elevated, and disappearing on pressure. It rapidly extends over the body, and reaches a maximum on the following day. The spots are apt to become confluent and form patches on certain parts, especially on the face. The eruption is attended with considerable irritation of the skin, and is followed by slight desquamation. The eyes are red, but there is little or no coryza ; the throat is almost always sore, and the glands in the neck may be a little swollen and tender. The amount of general disturbance is very trivial indeed as a rule, and the duration rarely extends beyond four or five days. Epidemic roseola differs from measles in the shortness of the pre-eruptive stage, the non-crescentic arrangement of the rash, the absence of coryza, the very slight fever, and the generally milder course. Previous attacks of measles exert no protective influence.

No treatment of a special kind is called for.

SMALL-POX—VARIOLA.

Small-pox is a specific disease caused by a poison which may be communicated by the emanations from the patient, or by inoculation with the contents of the pock. Hence, small-pox is both infectious and inoculable. The poison may be also conveyed to a distance by contaminated clothing, furniture, &c. Small-pox may be met with at any age, and affects those most severely who have not been previously vaccinated. A second attack is very unusual. Besides the eruption, which will be briefly described further on, there are *no characteristic anatomical changes.*

Symptoms.—The average duration of the stage of *incubation* is twelve days. The *invasion* begins suddenly with a well-marked rigor or repeated chills, accompanied by high fever, which rapidly rises to 103° or 104° (primary fever). The fever is more or less invariably associated with severe headache, pain across the loins, nausea and vomiting, in addition to the grave general disturbance common to high fever. With the exception of vomiting, which generally ceases towards the end of the second day, the foregoing symptoms increase in severity up to the appearance of the eruption. As occasional symptoms, we may meet with severe radiating pains in the limbs, convulsions or delirium. The latter are of very grave import, should they persist after the appearance of the eruption.

The *eruption* generally appears on the third day, and if later, is rarely, if ever confluent. It comes out first on the face, extending over the body in the course of one or two days. The number of the "pocks" varies much. Each begins as a small isolated congested spot; this enlarges, becomes indurated, and by the third day of the eruption, becomes a true *papule*, which feels like a small shot imbedded in the skin. Owing to the accumulation of serous fluid in the *rete mucosum*, the horny layer of the epidermis becomes elevated, and by the sixth day a perfect *vesicle* is formed. The vesicle is broad, flattened, and its cavity is subdivided by a network of altered epithelial cells. The latter fact, together with swelling and proliferation of cells at the periphery, probably accounts for the central depression of the vesicle known as umbilication. A red areola soon surrounds the vesicle, its contents become turbid, and finally purulent, its locular septa give way, forming a hemispherical *pustule*, which reaches its height about the ninth day. By these successive stages the period of *maturation* is reached. A diffused scarlet rash, not unlike that of scarlatina, and sometimes mistaken for it, occasionally precedes the typical eruption.

In all but the worst cases *the primary fever falls, and the initial symptoms subside on the appearance of the eruption.* In mild cases the remission is complete. But with the maturation of the eruption, other symptoms appear, the intensity of which is directly proportionate to the number of the pocks. Thus the temperature rises again, and in doing so, represents a systemic and suppurative fever akin to septicæmia (*Secondary Fever*). It continues in "discrete" cases for about seven or eight days. In addition to the ordinary febrile symptoms (which are marked in direct proportion to the intensity of the fever) the skin becomes greatly swollen, red, and very irritable, especially on the face. Since the "pocks" not infrequently appear on the various mucous tracts, sore throat, hoarseness, cough, diarrhœa, ophthalmia, or blindness may be observed. A peculiar and disagreeable odour is also apt to emanate from the patient.

The pustules now dry up (*desiccation*) and give place to dark coloured scabs which usually separate some time during the third week. Since the eruptive inflammation generally involves more or less destruction of "the true skin," cicatrices or "pits" occur. These are at first of a dull-reddish colour, but ultimately become quite white. With the subsidence of the secondary fever and its accompanying symptoms, convalescence rapidly takes place. It is at this time, however—especially in "confluent" cases—that serious complications are apt to arise.

VARIETIES.—Where the pocks are distinct and not numerous the disease is termed *discrete;* where they are at first distinct, but subsequently come into contact with each other—*coherent;* where they are numerous and run together—*confluent*. In *malignant* small-pox intense nervous depression, low muttering delirium, and general prostration are marked from the onset. Hæmorrhages under the skin, into the "pocks," and from the various mucous surfaces, are of frequent occurrence in malignant small-pox, and give rise to a *hæmorrhagic* sub-variety, which is especially serious and fatal.

COMPLICATIONS AND SEQUELÆ.—The most important are (1) *boils* on the skin, and *abscesses;* (2) *erysipelas* of the face and head; (3) *conjunctivitis* and *ulceration of the cornea*, sometimes leading to destruction of the eyeball; (4) *bronchitis*, and low forms of *pneumonia;* (5) *laryngitis*, sometimes followed by *œdema of the glottis*, dependent on the eruption within the larynx; and (6) *pyæmia.*

The *diagnosis* will be considered hereafter.

As to *prognosis*—very young children, and subjects above sixty years of age generally die. Pregnant women usually abort and die. It is especially dangerous in drunkards, in the unvaccinated, and in the confluent forms of the disease. Epidemic is more grave than sporadic small-pox.

VARIOLOID.—In those who have been vaccinated, the course of small-pox is so much modified, as to merit a separate description under the name of *varioloid.* Its main features are as follows:—1. The rash is scanty and ill-developed; (2) the development of the "pocks" is much more rapid, and often very imperfect; (3) there is either no "secondary fever" at all, or it is very slight, and only lasts for eighteen or twenty-four hours; (4) there is little or no "pitting;" and (5) the prognosis is almost always favourable.

TREATMENT.—In mild cases but little is required beyond isolation of the patient, rest in bed, cleanliness and good ventilation, a light nutritious and non-stimulating diet, and general attention to the excretions.

In graver cases we meet with important symptomatic indications. Thus during the stage of invasion, there may be great *nervous excitement* and *delirium*, which may be due either to general excitability, to the height of the fever, or to previous alcoholic excesses. In the *first* form, chloral, bromide of potash, and especially small doses of Dover's powder are required; in the *second*—cold spongings of the body, and the internal administration of salines with aconite or digitalis frequently repeated, and in the *third*—good nutritious food, with occasional subcutaneous injections of morphia. *Coma, nervous prostration, and low forms of delirium*, require a liberal allowance of stimulants and good food. In such cases I can also speak favourably of tepid sheet packs repeated from time to time.

As regards the *eruption*, protection of the surface from air and light by inunctions of different kinds, and keeping the room darkened are said to prevent "pitting." The troublesome *irritability of the skin* is best relieved by frequent spongings, followed by dredging with some absorbent powder, such as starch and oxide of zinc. During *desiccation*, baths should be taken, and the linen frequently changed. Complications must be treated as they arise.

VACCINIA—COW-POX.

Cow-pox is a specific contagious disease of cattle, which is characterised by a pustular eruption closely resembling that of small-pox, and appears in the cow on the udder and teats. It only occurs in the human being as the result of inoculation. The most recent investigations seem to show that it is essentially a distinct disease from small-pox, though when inoculated it affords immunity from small-pox; and like other specific diseases it confers protection against a second attack.

Methods of Inoculation.—*Source of lymph.*—It may be obtained direct from the cow; but for obvious reasons this source is rarely practicable. It is usually obtained from the human being, from whom it may be directly inoculated; or it may be collected in capillary tubes; or ivory points may be charged by dipping them in the lymph, and afterwards allowing them to dry. The advantage of either of the last two methods, is that the lymph may be preserved, and used as required. In whatever way the lymph is obtained, it should be taken from mature vesicles on the eighth day after inoculation, and unmixed with blood. *Time for inoculation.*—Vaccination should be carried out at two to three months of age, the child should be healthy at the time, and if unsuccessful it should be repeated. *Mode of introducing the lymph.*—The chief methods are *three* in number:—(*a*) by simple puncture with a lancet; (*b*) by tattooing; (*c*) by a series of scratches. The part usually selected for inoculation is the skin over the deltoid muscle of the shoulder. Liquid lymph direct from the vaccine vesicle, or from a capillary tube, should be inserted by the lancet; with dried lymph on ivory points, the latter should be introduced into the punctures or well rubbed on the surface previously abraded. Whatever method may be selected, the punctures or scratches should only penetrate the cuticle. If the deeper layers of the skin be wounded, hæmorrhage will occur, and inoculation will be uncertain. It is usual to inoculate at least four points on the arm, about $\frac{3}{4}$ inch apart.

Phenomena following Vaccination.—By the *third* day little red *papules* appear which become *vesicular* by the *sixth* day. The vesicles are roundish, flattened, and generally umbilicated. They are surrounded by a bright red inflammatory areola, and by the *eighth* day are at their height. The contained lymph is clear and slightly viscid, and it is only at this stage that it should be used for inoculation. On the *tenth* day the contents become purulent. Shortly after, the *pustule* begins to dry up, the inflammation subsides, and by the fourteenth or fifteenth day a hard dark scab has formed, which falls off about a week later, leaving a depressed pitted scar, which is permanent. These changes are associated with some general constitutional disturbance, and fever. The glands in the axilla often enlarge.

Re-vaccination is recommended about puberty, in order to confer additional protection from small-pox. In some cases no effect results; in others, the phenomena above related recur in the usual order, but the development is generally much more rapid, and the different stages are less marked.

The remote effect of successful vaccination is to confer absolute and complete protection against small-pox in the majority of cases; but where this is not obtained, the course of the disease is much modified, as described under the head of varioloid.

VARICELLA—CHICKEN-POX.

Chicken-pox is an acute specific fever, running a definite course, and communicable either by infection, or by inoculation. It never occurs twice in the same person, and is generally confined to children.

Symptoms.—The duration of the incubation stage of chicken-pox is most variable. The disease commences with slight fever and general malaise, which precede the eruption for about twenty-four or thirty-six hours. The eruption at first consists of reddish papules, which quickly become converted into largish single-chambered blebs or vesicles, filled with clear fluid. About twenty-four hours later the fluid becomes turbid, and the vesicles are surrounded by slight inflammatory areolæ. On the fourth or fifth day they rupture and dry up; and small scabs are formed, which come away gradually, leaving as a rule no discoloration or pitting behind. The rash usually appears first on the trunk, but soon extends to the face and other parts of the body. Fresh crops continue to come out for a few days. The general symptoms are slight and unimportant, but occasionally the convalescence is protracted.

The *prognosis* is invariably favourable.

No special *treatment* is called for.

ERYSIPELAS.

Erysipelas is an *acute specific disease*, characterised by a diffuse inflammation of the skin. It may be either *traumatic* or *idiopathic ;* that is to say, it may occur either in connection with wounds, or as a primary disorder ; but it is probable that they are essentially of the same nature. The latter form alone concerns the physician. From the fact that it is infectious (although to a less degree than the specific fevers already described), and is occasionally epidemic, it would appear that some *specific* poison, introduced from without, plays the part of an exciting cause. Erysipelas differs, however, from almost all other specific fevers, in the fact that one attack, so far from conferring protection against its repetition, renders the subject of it especially liable to its recurrence. Many cases, however, occur which it is impossible to trace to specific infection. General debility, by whatever cause induced, and insanitary surroundings, undoubtedly operate as *predisposing* causes. We have evidently much to learn yet with regard to the etiology of erysipelas.

Morbid Anatomy.—The skin becomes red, and infiltrated with serum and leucocytes, leading to various degrees of swelling. Where inflammation is more intense, *vesicles* and *bullæ* may form, containing clear or sanious fluid ; or subcutaneous suppuration of a diffuse kind may occur. The lymphatic glands and vessels of the part become enlarged and tender; and the coats of the veins may inflame, and lead to thrombosis. The inflammation may extend from the skin into the mucous orifices; *e.g.*, the ear, mouth, or nose.

SYMPTOMS.—The *local symptoms* are preceded by a period, lasting from three hours to two or three days, which is marked by chills, headache, digestive disturbance, rapid rise of temperature, and enlargement of the lymphatic glands of the part; and at the end of this premonitory stage the local signs appear. The skin becomes red, swollen, smooth, shining, painful, and tender at some point, starting from which, the process rapidly extends, and generally in some special direction. The line of its advance is well defined by an abrupt red margin, while the redness passes quite gradually into the healthy skin where the inflammation is subsiding. The amount of œdema and pain depends upon the nature of the cellular tissue; where this is dense, and unyielding, the swelling is comparatively slight, and the pain very severe; while the reverse is the case where the tissue is lax and abundant, as in the eyelids. The starting point of idiopathic erysipelas is generally on the face at the junction of skin and mucous membrane, especially the *nose*. Thence, it usually extends upwards over the scalp, and the swelling causes much disfigurement, even to obliteration of the features. In bad cases, vesicles and bullæ may form, and subcutaneous suppuration occasionally takes place. Erysipelas may also affect the throat or larynx, causing pain and difficulty in swallowing, or respiratory trouble.

The inflammation usually attains its height on the second or third day; but when the development is slow, it may be at its maximum in one part, while it is subsiding at another. As the swelling and redness abate, the vesicles and bullæ burst and scab over, free *desquamation* of the cuticle follows, and (when the scalp has been affected) the hair usually gets thin, or falls off.

General symptoms.—The temperature rises rapidly to a high grade, but with well-marked morning remissions. The pulse is frequent, full, and strong. More or less delirium is nearly always present, which is due to sympathetic excitement of the cortical layers of the brain, more often than to meningitis. The tongue is dry and brown. The appetite is lost, and the bowels are costive. In severe cases the *typhoid state* is apt to supervene. The urine is febrile, and a small quantity of *albumen* is frequently present, but chronic renal disease rarely follows. *Defervescence* usually occurs about the end of the first week, when temperature rapidly falls.

The more important *complications* are bronchitis, intestinal catarrh, pneumonia, meningitis, and œdema of the glottis.

The *prognosis* in uncomplicated cases is almost invariably favourable; except in very old people and infants, when it is often fatal.

TREATMENT.—Rest in bed, in a room of equable temperature, not too warm, and well ventilated, but free from draughts. Good nourishing food is required, and stimulants are frequently indicated at an early period. Full doses (♏ 20 to ♏ 40) of the tincture of the perchloride of iron, frequently repeated, are of great value. Quinine has also been recommended. Locally, the inflamed surface should be dredged over with flour, or starch with oxide of zinc, and covered with cotton wool. Such dressing should be bathed off daily with tepid water, and renewed. Shaving the scalp, followed by application of ice to the head, is of considerable service in bad cases with much delirium. In order to allay excitement, and to produce sleep, cold sponging, or wet cold packs, are useful when the fever runs high; while, as to medicines, bromide of potash and chloral hydrate are recommended in preference to opium.

GLANDERS—FARCY.

The above disorders can only be communicated to man by direct or indirect inoculation with the virus of a specific disease, which attacks the horse and animals of that class. Consequently, it is for the most part confined to grooms and stablemen. Glanders and farcy are mere varieties of one and the same disease; the former occurring as the result of poison introduced into the respiratory passages; while the latter depends on inoculation of some part of the trunk or limbs.

Morbid Anatomy.—The characteristic feature of the disease consists in peculiar nodular formations *(farcy buttons)* which vary in size from that of a pin's head to that of a hazel-nut, and appear on the mucous membrane of the nose, throat, mouth, &c., on the skin and subcutaneous tissues, or in various internal organs. They consist of cells, which rapidly soften and break down, forming ulcers when superficial, and abscesses when more deeply situated. The lymphatic glands are often enlarged.

Symptoms.—*Acute glanders*, after an incubation of variable length, sets in with rigors, fever, severe pains in the limbs, and great debility. Before very long, a copious and offensive discharge from the nose occurs; and about the same time a peculiar eruption appears on the skin, which is accompanied with fœtid sweats. The eruption consists at first of red spots, which soon become indurated and nodular. A pustule then forms over each spot, bursts, and leaves an irregular sloughy ulcer with a livid margin. Sores of a similar kind may occur in the mouth, throat, and air-passages; and abscesses are apt to form about the joints. There is also more or less erysipelatous redness of the nose, eyelids, and surrounding parts. Meanwhile, the patient becomes progressively weaker and more prostrate; and finally delirium and coma supervene, death usually taking place before the twentieth day.

Chronic glanders is rare. Its symptoms are on the whole analogous to those of acute form; but the eruption is often absent, and deep suppuration is more marked. Its course is very tedious.

Acute farcy usually depends on the inoculation of some part of the trunk or limbs. The lymphatics leading from the point of inoculation become inflamed, and there is enlargement of the neighbouring glands, followed by the formation of subcutaneous nodules and abscesses. There is, however, no discharge from the nose, and the eruption too is often absent. Farcy also occurs in a *chronic* form.

The *prognosis* is always very grave, but it is better in the chronic than in the acute forms, and in farcy than in glanders.

TREATMENT.—No specific is known. Stimulating, nutritious, and tonic plans of treatment should be carried out.

YELLOW FEVER.

THIS is a specific infectious fever, which confers protection against a second attack, and occurs almost exclusively in warm climates. A high temperature, in fact, is necessary for its production, and the disease is endemic in certain tropical regions. Filth, and insanitary conditions generally, appear to promote its occurrence.

Morbid Anatomy.—The liver is somewhat enlarged, and fatty. The mucous lining of the alimentary canal is injected and softened, and often more or less infiltrated with altered blood. The kidneys and spleen may be softened and degenerated. Hæmorrhages in various parts are common.

Symptoms.—With or without warning the invasion is sudden, and marked generally by rigors. The temperature rapidly rises to a variable height (101° to 105°), and is associated with the usual febrile symptoms. The most marked features of this stage are troublesome vomiting, constipation, tenderness of the epigastrium, intense frontal headache, redness and irritability of the eyes, with pains in the back and limbs. The urine generally contains albumen.

On the third or fourth day, the temperature falls and the symptoms subside; but the improvement is of short duration, except in very mild cases, when the disease may pass off altogether. In a few hours, though fever is slight or absent, the epigastric tenderness is increased, the vomit contains blood ("black vomit") and the stools are black from the same cause. Jaundice now usually supervenes, hæmorrhages from the mucous surfaces are apt to occur, and petechiæ may be observed. If the patient survives longer, he speedily passes into a state of collapse of a low "typhoid" type. The urine is scanty, or suppressed, and contains blood, albumen, and casts. Delirium, convulsions, or coma, are of common occurrence in severe cases.

Diagnosis.—Remittent fever may somewhat resemble yellow fever, but the former is not contagious, does not confer protection against a second attack, its febrile paroxysms are distinctly periodic, there is enlargement of the spleen, hæmorrhages are not so severe and persistent, and the disease is controlled by quinine.

The *prognosis* varies greatly in different epidemics. Death sometimes occurs in the early febrile stage from sudden collapse. In the second stage the gravity is proportionate to the prominence and persistence of hæmorrhagic and nervous symptoms.

TREATMENT.—Quinine has no specific action. The food should be light, liquid, and nutritious, and alcoholic stimulants are of great value. If these cannot be retained on the stomach they should be given *per rectum*. The *vomiting* may be met with ice internally, hydrocyanic acid, creosote, or chloroform; and in such cases, food should be given in very small quantities, frequently repeated. Opiates should be used with caution. The wet sheet-pack has been recommended; and the internal administration of carbolic acid, or the sulphocarbolates, has been very favourably spoken of.

CEREBRO-SPINAL FEVER.

CEREBRO-SPINAL fever is an acute specific disease, which is characterised by inflammation of the membranes of the brain and spinal cord. It is both *epidemic* and *infectious*, but little or nothing is known as to the conditions which determine its appearance or propagation. Young persons are most liable to be attacked.

Its *morbid anatomy* consists in inflammation of the pia mater of the brain and cord, attended with a purulent or semi-purulent exudation into the sub-arachnoid tissue, and an excess of somewhat turbid fluid in the ventricles. The inflammation may be either generally distributed, or limited to certain parts of the surface of the brain or cord. The substance of the brain and cord is congested.

SYMPTOMS.—The disease usually *commences very abruptly* with a violent chill, intense headache, and persistent vomiting. Stiffness and retraction of the neck, pain down the spine and radiating to the limbs (much increased on movement), and general cutaneous hyperæsthesia are also early and characteristic symptoms. The head is usually retracted, and there may be more or less tonic contraction of the extensor muscles of the neck and spine. There is almost always a certain amount of delirium, often of a violent type, and sometimes alternating with insensibility. In very mild cases, however, the patient may be merely dull, drowsy, and restless. The face is pale and the expression is anxious. The pyrexia, though variable, is usually moderate in amount, and runs a most irregular course.

The disease now rapidly develops to its full intensity. About the second to the fifth day various cutaneous eruptions may appear. Of these, *herpes* on the face and lips is the most common, but petechiæ, small patches of erythema, or roseola, are not infrequently met with. There is as a rule more or less intolerance of light and sound; and ptosis, strabismus, or irregularity of the pupils may be sometimes observed. Tinnitus and partial deafness are also not uncommon. The pulse is feeble, and liable to great variations in frequency; the tongue is brown and dry; the appetite fails, and the bowels are constipated.

In fatal cases the symptoms of irritation subside, and are replaced by those of general depression; death taking place in a few days from coma. In favourable cases the signs of depression are either absent, or comparatively slight. The symptoms gradually subside, and convalescence occurs in one or two weeks; but it may be protracted for a long time, with pain in the back and disturbance of the sight or hearing.

The most important *sequelæ* are deafness, derangements of vision, chronic hydrocephalus, and chronic meningitis. The mortality varies within such wide limits as from 20 to 80 per cent.

Treatment.—The most important indications in the acute stage are—rest in bed, cold applications to the head, local abstraction of blood by leeches placed behind the ear, and the free and early hypodermic injection of morphia. In the later stages, iodide of potassium combined with tonics appears to be of some service.

INFLUENZA.

Influenza is an *epidemic* form of catarrh, chiefly affecting the respiratory and digestive tracts. It is apparently dependent upon a *specific poison*, the nature of which is unknown; and it is usually regarded as *infectious*. A weak state of health, insalubrious surroundings, and overcrowding constitute *predisposing causes*.

The *anatomical characters* are those of ordinary catarrh.

Symptoms.—After an incubation period of uncertain length, the disease usually sets in suddenly, with chilliness, lassitude, pains in the limbs, and frontal headache, followed by high fever, thirst, great prostration, and debility. *Locally*—smarting of the eyes, a watery acrid discharge from the nose with sneezing, occasional epistaxis, sore throat, deafness, hoarseness, and cough with mucoid sputa, indicate a progressive catarrh of the respiratory tract. When the digestive tract is affected, the tongue is furred, and there is also anorexia, epigastric pain and tenderness, nausea and vomiting, and diarrhœa. The *urine* is febrile. In uncomplicated cases, the duration does not usually extend beyond four or five days, but convalescence is often protracted, and is characterised by marked debility and general prostration, and often by troublesome cough, or neuralgic pains. In grave cases, there may be delirium, convulsions, and coma; or the "typhoid" state may supervene. Capillary bronchitis and pneumonia are common complications. Under such circumstances the duration is longer. Though the prognosis is favourable as a rule, still it may prove fatal with the aged and feeble.

Treatment.—No lowering treatment is admissible. The diet should be nutritious and liberal, regulated according to the digestive capacity. Alcohol is necessary in a large proportion of cases, especially among the aged and feeble. Medicinally, quinine (gr. i.-ij.), salicine (gr. x.-xv.), or antipyrin (gr. v.-x.), may often be given every four or six hours with advantage. An opiate at bed-time for the first few nights, in a dose just sufficient to relieve pain and restlessness, is frequently most beneficial. The sneezing and frontal headache are often relieved by liq. arsenicalis (♏ ½ every two or three hours), or by sponging the face with water as hot as can be borne. Inhalations of steam medicated with conium or chloroform, stimulating expectorants and poultices, fomentations or sinapisms to the chest, may be employed to allay the catarrhal symptoms. During convalescence, tonics such as quinine and iron are indicated.

DIPHTHERIA.

Diphtheria is an acute specific disease, epidemic and contagious, and is characterised by more or less profound general disturbance, with inflammation of the throat and adjacent parts. The affection is not only directly communicable by contamination with diphtheritic products, and exhalations from the patient, but it may be indirectly conveyed by infected clothing and other external objects. There would also appear to be some definite relation to insanitary surroundings.

Diphtheria is very apt to occur for a long time about the locality in which it occurs. The young, feeble, and nervous are especially liable to be attacked, and girls more so than boys.

The term "diphtheritic," as pertaining to this disease now under consideration, must not be confounded with the term applied to a morbid-anatomical condition of mucous membrane (*vide* p. 10). In the latter sense, it refers only to the *degree* and *intensity* of inflammatory change, and not to the circumstances of its origin; though the condition is most commonly found in connection with diphtheria. On the other hand, diphtheria, in its typical form, is attended with such severe and intense throat-inflammation, that the local condition is usually characterised by diphtheritic "false membrane." This association, however, is not constant; and in mild cases the throat-mischief may be of a catarrhal or croupous nature. In practice we often meet with what are called "*low* sore-throats" unaccompanied by false-membrane; which, if not instances of mild diphtheria, are closely related and strictly analogous to it.

Morbid Anatomy.—The inflammation usually begins in the throat, affecting the tonsils, soft palate, or pharynx, or all of these parts. Thence it may spread to the nose, larynx, and air-passages (even to the remote bronchi), or more rarely to the alimentary canal. The mucous membrane is at first red, swollen, and secretes viscid mucus. The inflammation extends, and at one or more points an exudation appears, patches are formed, and ultimately the entire surface of the throat may be thus covered with what is called "false membrane." The latter is at first quite thin and of a greyish-white colour, and is removable from the surface on which it lies. In mild cases it may not progress beyond this point. More often, however, it involves deeper parts, it increases in thickness, becomes of a dirty yellow colour (like chamois leather), and can no longer be detached without injury to the mucous membrane which it infiltrates. The false membrane is usually thinner, softer, and less firmly adherent, the farther it gets from the point of origin. Microscopically, it consists of fibrin, in which cells, nuclei and granules are imbedded. Later on, the false membranes disintegrate and come away, leaving either superficial erosions, or well-marked ulcers, which (in favourable cases) finally cicatrise. Hæmorrhage from the inflamed surface is not uncommon, and blood then mingles with the exudation. Rod-shaped bacteria, and micrococci are also found in the exudation, but their relation to the disease is not yet definitely ascertained.

Symptoms.—The onset of diphtheria sometimes begins abruptly with a severe rigor and vomiting; but more often it is gradual, and marked by chilliness, depression, loss of appetite and general malaise, which are soon followed by more or less pyrexia, soreness of the throat, stiffness of the neck, painful if not difficult deglutition, and slight swelling of the glands at the angle of the jaw. The throat at this stage is red and swollen, with one or more small greyish-white or yellowish spots on the tonsil, soft palate, or back of pharynx; or if the nasal cavity be already implicated, one or other nostril may be obstructed. It is well to remember that the severity of the early symptoms is no safe criterion of the gravity of the case.

When the disease becomes fully established all the above symptoms grow much worse. Deglutition is painful, difficult, or impossible. The breath is foul, the breathing is impeded, the voice is muffled or nasal in tone, and the glandular enlargement of the neck increases but has no tendency to suppurate. The condition of the throat has already been alluded to. The fever is rarely high, but is attended with intense prostration, headache, restlessness, and perhaps vomiting; the tongue becomes dry and brown, the skin pale, cold, and clammy; the pulse rapid, feeble, and irregular; and delirium if present is usually slight and nocturnal. The urine is almost always *albuminous* from the onset.

Diphtheria not only varies widely in severity, but the symptoms differ according to the part which is affected. If the nares be involved, there is more or less obstruction of one or both nostrils, with a fœtid discoloured discharge. If the larynx and air-passages be implicated, the symptoms are those of laryngeal croup. Death occurs in more than half the cases, and may be due to asphyxia (chiefly during the first week); or to asthenia (chiefly during second week); or to syncope (sometimes occurring quite unexpectedly). In favourable cases gradual recovery usually takes place in the second week.

Apart from the low inflammatory complications which sometimes occur, we not infrequently meet with peculiar nervous sequelæ of a paralytic nature (even after the mildest attacks) which usually come on during the second or third week of apparent convalescence. Thus, there may be paralysis of the soft palate and pharynx, causing a nasal tone of voice, regurgitation of fluids through the nose, and difficulty of swallowing; of the ocular muscles, causing squint; of the limbs (especially the legs), causing more or less loss of power. Some disturbance or impairment of sensation usually accompanies the paralysis. The pathology of such paralyses is obscure, but evidence points to their dependence upon diffuse neuritis.

Treatment.—Rest in bed, pure air, alcohol in some form, and good nourishment are urgently needed from the outset. Beef-tea (strong) and milk should be given at short intervals; and if from any cause food cannot be taken by the mouth, it should be given *per rectum*. Stimulants should be employed freely. To the youngest child a teaspoonful of brandy or whisky every two hours may often be given with advantage. If brandy cannot be retained, champagne is a good substitute. As to medicine, nothing is better than full doses of the tincture of the perchloride of iron, combined with chlorate of potash, and dilute hydrochloric acid, to be given every three or four hours. As to local treatment, some advise swabbing out the throat with a solution of nitrate of silver (gr. xx. to ʒ j. of water), or equal parts of strong hydrochloric acid and honey. If an application of this kind be resorted to, it should be made once for all at the outset of the throat mischief, and should not be repeated. Washes or sprays of solutions of borax (saturated), carbolic acid (5 per cent.), permanganate of potash ($\frac{1}{2}$ per cent.), chlorate of potash (2 per cent.), or sulphurous acid are preferred by the writer. Great comfort is also derived from sucking ice constantly and freely. Emetics are sometimes useful as a means of inducing expulsion of false membranes from the larynx. Sleeplessness should be combated with an opiate at night, which is usually well borne. During convalescence, quinine, iron, and change of air are required. The nervous sequelæ are best treated by strychnia, galvanism, and the application of blisters alongside the spinal column.

EPIDEMIC CHOLERA.

Cholera is an *epidemic* disease, sometimes *endemic*, and dependent on a *specific poison.* As to the nature of the latter our knowledge is very limited. It briefly amounts to this: that it exhibits the characters of an organised contagium (comma bacillus?) which is present in the rice-water stools of those suffering from cholera; that the discharges when quite fresh are inoperative, but become virulently infective in the course of from two to five days, after which they lose their poisonous influence.

That the poison enters the body through the alimentary canal, to which it is generally conveyed by infected drinking water; that it is rarely if ever directly conveyed through the air by emanations from an infected person; and that its operations are favoured by obscure meteorological conditions, by insanitation, and by individual debility.

Anatomical Characters.—When death occurs during the stage of collapse, the body is shrivelled and shrunken, and the dependent parts are congested. Rigor mortis is well-marked and prolonged. The blood is thicker and darker than usual; the pulmonary artery, right side of the heart and systemic veins are generally gorged with blood, while the left side of the heart is nearly empty. The spleen is small. The intestinal mucous membrane has a sodden appearance and its follicles are enlarged and prominent. The contents of the bowels resemble the discharges during life, added to which there is abundance of shed epithelium (probably a post-mortem change). The large bowel is usually contracted.

If death occurs during reaction, the tissues are moister, and the blood is more equally distributed.

Various secondary inflammations are met with as complications.

SYMPTOMS.—After an *incubation* of uncertain length, there are usually some premonitory symptoms for a day or so, characterised by malaise, depression, and slight diarrhœa, but the onset is sometimes sudden.

1. *Evacuation stage.—Purging* (with or without pain) is the first symptom. The *stools* are watery and profuse, at first fæcal, but soon become thin, neutral or alkaline, colourless, and almost odourless, with whitish flakes in suspension (*rice-water stools*). The quantity of fluid thus discharged is often enormous.

Vomiting comes on a little later. The vomit is at first bile-stained, but soon resembles the rice-water stools and is very abundant. Painful *cramps* in the limbs and abdomen, and *intense thirst* soon follow, culminating in the collapse which is characteristic of the next stage.

2. The "*algide*" *stage* is marked by profound *collapse*. The *tissues* shrink, the features are pinched, the eyeballs become sunken, and the cheeks hollow. The *surface* is more or less livid, cold and clammy. The *surface temperature* falls to 94° or 95° or lower, while in the rectum it may be as high as 101° or more. The *pulse* is small, rapid and almost imperceptible; the *respirations* are rapid and shallow; the expired air is cool; and the *voice* is fainting and whispering. The *urinary* and *biliary secretions* are totally suppressed. The patient throws his arms about, and becomes restless and wakeful, and singularly apathetic. He complains of intense *thirst* and heat at the epigastrium. Finally, the vomiting and purging cease, or are greatly diminished, and the patient lies helpless as a corpse. The *mind* is clear at first, but in fatal cases *stupor* sets in, followed by *coma*.

The *duration* of this stage varies from two to twenty-four hours, and it also varies much in intensity. In severe cases the symptoms are well marked, and death occurs from coma, most often between the tenth and fourteenth hours. In more favourable cases reaction may take place.

3. The *stage of reaction* sets in gradually with general improvement, the pulse becomes stronger, the respiration slower, and the skin loses its livid and shrunken aspect. The thirst, diarrhœa and restlessness subside; the secretion of urine returns, and the stools are once more stained with bile. The body temperature is more equally distributed—that of the surface rises, while that of internal parts falls. The patient may fall asleep, and recovery may be re-established in the course of twelve to twenty-four hours. More often, however, the reaction assumes a febrile type, and is more severe and prolonged ("cholera-typhoid").

Even now recovery is not assured, and death may occur from complications arising during the stage of reaction, such as suppression of urine, inflammation of the bowels, low form of pneumonia, cholera-typhoid, or cardiac thrombosis.

Variations.—Cholera sometimes sets in suddenly with no premonitory symptoms, and death occurs speedily from collapse. The term "choleraic diarrhœa," or "cholerine," is sometimes applied to those cases occurring during an epidemic of cholera which are characterised by milder symptoms and far less mortality. Some again recognise a relationship between epidemic cholera and bad cases of summer diarrhœa (English cholera).

The *diagnosis* of true cholera rests upon its epidemic character, the abundant rice-water stools, the rapid tendency to prostration and collapse, and the high mortality.

The *prognosis* is necessarily very grave; and the mortality is on the average about fifty per cent.

Treatment.—It is impossible to over-estimate the importance of *preventive treatment*, by careful attention to purity of drinking water, strict sanitation, domestic cleanliness, and immediate disinfection of all evacuations and contaminated articles. All diarrhœas during an epidemic should receive prompt attention.

During the stages of evacuation and collapse, the patient should rest in bed, and take nothing but ice to suck; a large mustard poultice should be applied to the abdomen; and frictions and hot bottles may be used to maintain warmth. Medicinally, opium may be given in grain doses in combination with two or three grains of acetate of lead; or, if the former be rejected, small doses of morphia may be injected subcutaneously. When the purging and vomiting are severe it is worse than useless to attempt to give anything beyond the ice; but if it can be retained, a little beef-tea, chicken-broth, or milk, may be given either by the mouth or the rectum.

When *reaction* sets in the diet should be very bland and light, and only increased and improved with great care. Stimulants are not necessarily required, but should the "typhoid state" supervene, they are beneficial. Water may be freely allowed, to which the addition of salt and carbonate of soda is recommended. Complications and sequelæ must be treated as they arise. Tonics and preparations of iron are often serviceable during convalescence.

Innumerable *specifics* have from time to time been advocated, but have not justified the expectations formed of them.

DYSENTERY.

It is not yet settled whether dysentery is a *specific* disease or not, but the evidence is mainly on the affirmative side. Dysentery occurs both in an *epidemic*, and in a *sporadic* or scattered form. Both forms are much more frequent in tropical, than in temperate climates. Foul water, polluted air, insufficient food, exposure, and fatigue favour the occurrence of dysentery, but we have not yet succeeded in isolating the exciting cause of the disease. Like cholera, it is probably not directly infectious, but arises from the introduction of infective matter into the alimentary canal.

Morbid Anatomy.—The essential feature consists in an inflammation of the mucous membrane of the colon, especially towards its lower end. The small intestine is rarely implicated. The local appearance chiefly depends on the severity of the inflammatory process. In mild cases there are patches of catarrh, characterised as usual by congestion and swelling of the mucous membrane, mucous exudation, and cellular proliferation. The solitary follicles are unduly large and prominent. Superficial erosions or follicular ulcers may occur here and there, the peritoneal aspect of which may exhibit signs of slight inflammation. In severe cases the inflammation is of the diphtheritic type, and is characterised by patches of fibrinous exudation on the surface and into the substance of the mucous membrane, which is followed by necrosis and ulceration. As a rule only the mucous membrane is involved, but the slough sometimes includes the deeper tissues and may thus lead to hæmorrhage or perforation. In favourable cases the ulcers ultimately cicatrise, but the process is liable to be followed by puckering and more or less contraction of the intestine. In other cases a chronic inflammatory condition is left behind, attended with a muco-purulent discharge.

Tropical abscesses of the liver, enlargement and softening of the mesenteric glands, bronchitis, pneumonia,

&c., are not infrequently met with in association with dysentery.

Symptoms.—The symptoms of acute dysentery—like the morbid changes—exhibit every variety, from a mild sporadic case to an epidemic of the most virulent type.

The following description applies to a moderately severe case. Irregular action of the bowels, griping pains, lassitude, and anorexia, generally precede the special symptoms for a few days. The actual attack is ushered in by shivering or rigors with fever. Griping pains (*tormina*) are felt in the abdomen, with a strong desire to evacuate the bowels. The stools are at first fæcal and abundant; but they soon lose this character, becoming scanty, bloody, and slimy from the presence of mucus, and emit a most offensive odour. Still later, they become thin, watery, and dirty-looking, and perhaps contain fragments of sloughy mucous membrane, or dark-coloured particles of blood. Profuse hæmorrhage sometimes occurs about this period. The desire to defæcate now becomes almost constant, and is attended with much tenesmus, irritation of the anus, and sometimes with very frequent micturition and dysuria. The tongue is furred, and complaint is made of thirst, loss of appetite, and occasionally of nausea and vomiting. The abdomen is tender, but not markedly tympanitic. There is always more or less febrile disturbance of a depressing type, which varies in amount and duration, according to the extent and severity of the local mischief.

In favourable cases improvement begins about the end of the first week, but the convalescence is usually protracted. When a fatal termination threatens, the symptoms assume an adynamic or typhoid character, with great prostration, a dry brown tongue, hiccup and vomiting, and more or less tympanitis; the patient finally becoming comatose. In the event of hæmorrhage, perforation, or the formation of abscesses in the liver, the symptoms preceding death will vary accordingly.

In other cases the disease may become *chronic* and continue for an indefinite time. The action of the bowels is irregular; the stools are offensive, and present from time to time a mucous, bloody, or serous appearance, according to circumstances. The general health suffers, as indicated by anæmia, loss of flesh, with a sallow and aged expression.

Diagnosis.—The symptoms and the characters of the stools as described are quite characteristic.

Prognosis.—Sporadic dysentery is not nearly so fatal as the epidemic form. Much depends too on the type of the epidemic, the general condition of the patient, the presence or absence of complications, and the mode of treatment adopted. Very chronic cases are extremely troublesome.

Treatment.—In the first place it is necessary to remove the patient to cool, clean, and healthy quarters; rest in bed being enjoined. In mild cases it is well to begin the treatment with a dose of castor oil to empty the bowels, and sometimes small doses of the oil (♏v.-x.) combined with laudanum may be continued afterwards with good effects. In more severe cases preliminary purgation should be omitted, or employed with great caution. At the outset, hot fomentations or ten to twenty leeches may be applied to the abdomen, to relieve local pain and tenderness. Opium should be given, either by the mouth or in a small enema of starch, in sufficient quantity to relieve pain and tenesmus.

Ipecacuanha (with or without opium) has been strongly recommended, in doses of gr. xxv.-xxx. with a little syrup of orange. If absolute rest in the recumbent posture be observed, and no fluid (beyond perhaps a little ice to relieve thirst) be taken for some three hours after, sickness rarely follows. It should be repeated at intervals of eight to ten hours according to the urgency of the case. The diet should consist of milk, gruel, broth, eggs, soups, and the like, with stimulants in accordance with the usual indications for alcohol.

In the *chronic* form ipecacuanha is often useful in doses of about gr. v. thrice daily. Astringents and tonics are also of great value, especially iron in full doses. Enemata containing copper, lead, nitrate of silver (gr. ½ ad ℥ j.), or sulphate of zinc have been recommended in obstinate cases. A flannel bandage round the belly should be worn, and cold or tepid bathing with friction is often desirable.

HYDROPHOBIA.

Hydrophobia is an acute specific disease, exclusively induced by direct inoculation from an animal afflicted with "rabies" or "madness," usually from the bite of a mad dog or cat. The poison is probably conveyed by the saliva of the animal.

The original wound generally heals readily, and the actual disease only develops after an interval of incubation, which is remarkable in its long and uncertain duration. The usual length of this period is from one to two months; but it has been known to vary from three or four weeks to as many months or even years. Neither sex or age has any influence in its occurrence.

The disease begins with general malaise, depression of spirits, sleeplessness, and more or less restless anxiety. There may be uncomfortable sensations or actual pain at the seat of the primary wound, but it is a purely subjective condition, and is probably induced by persistent attention to the part. The first really characteristic symptom is a sense of discomfort in the throat, with some difficulty in swallowing liquids, from interruption by a short sharp inspiratory gasp, resembling (as Dr. Gowers says) that caused by a sudden affusion of cold water. This respiratory spasm soon becomes more violent, till it involves all the extraordinary muscles of inspiration. The angles of the mouth are drawn outward, giving a curious sardonic expression. The spasm also becomes more readily excited, till at length it is induced by any sudden external impression, such as the mere sight of water, a draught of cold air, a loud noise, &c. The saliva cannot be swallowed, and the patient tosses about and spits wildly to get rid of it.

From this point the disease quickly develops. On the one hand, the spasmodic tendency becomes more and more widely distributed, until general tetanoid convulsions are induced over and above the respiratory spasm, under the influence of stimuli such as those already mentioned. On the other hand, the general distress deepens, merging into delirium, or maniacal frenzy, with delusions and hallucinations. The face is flushed, there is moderate fever, little or no food can be taken, vomiting is not uncommon, and the patient quickly passes into a state of profound exhaustion. In some cases the symptoms may exhibit a remarkable abatement towards the last, but the fact affords no justification of more hopeful prognosis.

The usual duration is from one to four days. Death is almost invariable, and generally occurs from asthenia. Sometimes, however, it occurs suddenly during a paroxysm—from asphyxia. With regard to *diagnosis*, the one characteristic symptom is respiratory spasm, induced by attempts to swallow, and increasing in violence until it merges into convulsion.

The morbid anatomy is not characteristic; though Dr. Gowers attaches much importance to the following condition:—"Accumulation of leucocytes around the vessels, and their infiltration into the adjacent tissue; this change being either confined to, or most intense in, the medulla, contiguous to the lower part of the fourth ventricle."

Treatment by medicinal remedies is practically hopeless. In recent years, the method of inoculation adopted by M. Pasteur has attracted much attention; and, though not free from danger in itself, has saved some —perhaps many—lives.

Preventive measures are important; and if practised immediately, or soon after a bite from a rabid animal, much may be done. A tight ligature should, if possible, be at once applied above the wound, and the latter should then be well washed with warm soap and water, and afterwards thoroughly treated with some disinfectant fluid or caustic (nitrate of silver or nitric acid), or it should be excised.

SEPTICÆMIA—PYÆMIA.

The above terms are used to denote blood-poisoning resulting from the absorption of putrefactive material into the body. Various morbid conditions are probably included under this head, but our present knowledge of the subject is not precise enough for their exhaustive distinction and classification. A primary distinction must, however, be drawn between the two following conditions. In the one, the poison does not multiply in the blood, the effects are in direct proportion to the amount absorbed, and the disease thus caused is incapable of spreading by infection. In the other, the poison multiplies in the blood, produces effects out of proportion to the quantity first introduced, gives rise to special lesions, and is communicable by infection. The one then is a putrid intoxication, the other is a putrid infection. Putrid intoxication may exist alone, but it is difficult to suppose that putrid infection ever exists without more or less putrid intoxication. To avoid confusion, it would be well to denote all toxic cases, pure and simple, by the term Septicæmia; and all infective cases by the term Pyæmia. In actual practice, however, it must be admitted that this distinction is not always practicable. In each, the special poison is the result of a form of putrefactive decomposition, in which the agency of bacteria is essential. These bacteria are incapable of producing the poison in healthy tissues, and in the vast majority of cases the poison originates in a local lesion followed by inflammation, especially when the latter is in direct communication with the external air. Impure air (by over-crowding or otherwise) the puerperal condition, intemperance, and chronic disease of eliminating organs, are among the more important predisposing causes. The present state of our knowledge does not admit of exactly specifying the nature of the putrefactive processes on which these affections depend; but in Septicæmia it would appear that the *septic products* of bacterial activity are alone absorbed; while in Pyæmia the whole system becomes infested, in addition, with micro-organisms which actively multiply within the body.

SEPTICÆMIA.—The *post-mortem* appearances are not very striking. Decomposition sets in more rapidly than usual. The blood is dark, and coagulates imperfectly; and the internal organs are congested and softened.

Clinically, the disease is generally ushered in with chilliness followed by fever of an irregular type, accompanied with general depression and muscular prostration, loss of appetite, nausea and vomiting, irregularity of the bowels, headache, and occasional delirium of a low type. The duration varies greatly. In grave cases, the tendency is to death, preceded by stupor and collapse.

Pyæmia.—This affection was formerly supposed to depend upon the presence of pus in the blood; hence the term "pyæmia." Suppuration—it is true—may be the starting point of pyæmia, but only under special circumstances of putrefaction. Pyæmia is a specific infective condition, which arises secondarily to unhealthy necrotic change going on in some part of the body. It usually occurs in connection with an open wound, or at least some breach of tissue, through which the poison finds entrance into the system. It may follow traumatic injury of any kind, parturition, ulceration, and other forms of inflammatory mischief. Beyond the fact that pyæmia may be conveyed directly from affected persons, and furthered by dirty and insanitary surroundings, we know very little of the circumstances which determine its occurrence.

A characteristic feature of the morbid anatomy of pyæmia is formation of secondary or metastatic abscesses. They are found most frequently in the lungs, but they also often occur in the liver, spleen, kidneys, brain, and other organs. They are undoubtedly of embolic origin, and the suppurative changes which take place must be regarded as clear evidence of the infective nature of the embolus. Suppurative inflammation, moreover, is apt to occur in the subcutaneous tissue, and in the serous cavities of the joints, pleura, and pericardium; especially in the more chronic forms of the affection. These secondary lesions abound in micro-organisms (staphylo- and strepto-cocci).

The symptoms of Pyæmia are characterised by repeated well-marked rigors, accompanied with high fever, and followed by profuse sweating. The indications of general disturbance are much the same as those already described under the head of Septicæmia, but are more severe. More or less congestion of the lungs is generally present, and thus cough, expectoration, with pains in the chest are common symptoms. Special symptoms also occur, which are directly attributable to metastatic suppuration, and will thus vary according to circumstances. The prognosis is extremely unfavourable, and death occurs either from asthenia or some secondary visceral complication.

Pyæmia is not always of this acute nature. Sometimes it runs a chronic course, the symptoms are less marked, and the fever is of the intermittent type. This fact should especially be borne in mind, in connection with a pyæmic condition of great interest to the physician, arising from a putrid infective form of ulcerative endocarditis, of which something will be said in another place.

The indications for the treatment of these conditions are few and of a very general kind. When the lesion constituting the source of infection, can be got at, surgical measures should be adopted to reduce the putrefactive process, and to promote a more healthy condition of the part. The patient must be well supported by nutritious food and stimulants; while the various excretions should be encouraged. Large doses of quinine and salicylic acid have been recommended, on account of their supposed antiseptic effects. Septicæmia and Pyæmia are to a large extent preventable diseases; and of late years, with a better knowledge of sanitation, and improved methods of surgical treatment, there has been a very great reduction in the frequency of their occurrence.

MALARIAL FEVERS.

Such is the term applied to acute specific fevers produced by *marsh-poison* or *malaria.*

Nothing is known respecting the intimate nature of malaria. The conditions necessary for its production, are (*a*) a certain porosity of the soil; (*b*) a certain degree of saturation of the soil with water; and (*c*) a certain elevation of temperature. To what extent decay of vegetable matter operates—if at all—in the production of malarial disease, is uncertain; but the latter is undoubtedly most common in low-lying marshy ground. The poison is generated more actively by night than by day, hanging over the ground in a sort of thin mist, and extending to no great distance above it; hence, in such districts it is safer to sleep in the upper storeys than on the ground floor. It may be carried for considerable distances by the wind, but its progress is arrested by intervening obstacles, such as a range of hills, or a belt of trees; and it is absorbed by passing over any broad surface of water.

Malarial fevers are strictly *endemic*, and are not contagious, neither are they protective, but on the other hand render the subjects of them especially liable to their recurrence. They are more common in the autumn season.

Two well-marked forms are recognised, viz.—*Intermittent fever*, or *ague*, and *remittent fever.* Intermediate varieties not conforming exactly to either one type or the other are of frequent occurrence.

Morbid Anatomy.—The spleen is enlarged from congestion. The enlargement at first subsides during the intervals, but later on it becomes permanent from hyperplasia of the splenic substance. The blood contains more or less black pigment, and the blood-corpuscles are diminished in number. The liver, lymphatic glands, and intestinal mucous membrane are congested and softened.

I. Intermittent Fever—Ague.

Symptoms.—Ague is characterised by febrile attacks of regular periodic occurrence, separated by intervals of complete apyrexia. These attacks either come on suddenly, or are preceded for some few days by general malaise.

The actual attack of ague comprises three distinct stages—a cold, a hot, and a sweating stage.

1. The *cold stage* begins with shivering, which soon passes into a severe rigor. The patient feels cold, and his teeth chatter. The skin is dry and pale, the breathing is hurried, the pulse is rapid, small, and irregular, and he complains of headache, pains in the back and limbs, with loss of appetite. The urine is pale, abundant, and is passed frequently. During the *whole* of this stage the temperature rises rapidly, reaching a high grade. The duration varies greatly, but the average is from half an hour to one or two hours.

2. The *hot stage* as a rule sets in gradually, and the feeling of cold first gives place to one of pleasant warmth, but shortly afterwards the heat becomes intense. The skin is dry and pungently hot, the breathing is more rapid and deeper, while the pulse is full, tense, and frequent. The patient is excited and flushed, the thirst and headache are extreme, and sometimes there is more or less delirium. The urine becomes

darker and of higher specific gravity, and is intensely acid. The high temperature is maintained. The average duration is from one to four or five hours, but is often longer.

3. *Sweating stage.* Profuse perspiration now sets in, and appears first on the face; a general sense of comfort supervenes, and the above symptoms subside. The quantity of urine diminishes, and it deposits urates on standing. The temperature rapidly falls, the patient drops into a refreshing sleep, and wakes well. The duration is very variable.

The period intervening between one attack and the next is called the *intermission* or *interval*. During this time the health is usually good, but occasionally there is more or less malaise. The temperature is now normal.

Certain *varieties* are distinguished according to the length of the interval. Of these the more important are the *quotidian*, in which the attacks occur every 24 hours; *tertian*, every other day; *quartan*, every third day. In the quotidian form the attacks are the earliest, and last the longest of the three; in the quartan the reverse obtains; and the tertian form occupies an intermediate place.

II. Remittent Fever.

This form of malarial fever is almost confined to tropical climates. It is closely analogous to the intermittent form, but the essential difference lies in the fact that, during the interval the fever only *remits*, and does not entirely disappear.

The *symptoms* differ somewhat from ague in their mode of development. Thus, the *cold stage* is of shorter duration, and sometimes escapes recognition. The *hot stage* is much prolonged and continues for 6 to 12 hours or more. Gastro-intestinal and nervous symptoms are especially well marked. Vomiting is severe, and the vomited matters sometimes contain blood. The tongue is dry and parched; and occasionally there is slight jaundice. Headache is constant and distressing, and sometimes ushers in delirium and coma. The *sweating stage* is imperfect, and passes imperceptibly into the period of remission. The attack sometimes assumes an adynamic or "typhoid" type.

These attacks usually recur about noon each day, for 5 to 14 days, with an imperfect remission each morning. More rarely there are two attacks daily. In favourable cases the severity of successive attacks gradually decreases; and the prognosis is good in proportion to the distinctness and length of the remission, and to the absence of complications. Speaking generally, remittent fever is far more serious than the intermittent type.

Repeated attacks of malarial fever are apt to induce a depraved condition of the general health, which is described under the name of "malarial cachexia." It is characterised by a sallow complexion, weakness, and various functional disorders, such as dyspepsia, neuralgia, or pains in the muscles and joints. Hæmorrhages are also sometimes met with. The spleen is usually enlarged, hard and tender *(ague-cake)*; and in some cases the liver is also increased in size.

TREATMENT OF MALARIAL FEVERS.—*Preventive.* Those who live in malarial districts should avoid going out at night, or in the early morning; should not sleep on the ground floor; should not drink the water of the district, unless previously boiled and filtered; and finally when attacked should remove (if possible) to some healthier locality without delay.

During the paroxysm, the obvious indication in the cold stage is the application of warmth, while in the hot stage the body should be kept cool by sponging the skin with tepid water, free ventilation, and light clothing. Cooling drinks may also be given, and a little opium if there be great restlessness.

During the intervals, quinine and arsenic are our sheet-anchors, and unquestionably exert a powerful influence over the malarial poison. If quinine be employed (and in most cases it is preferable to arsenic), it must be given in large doses of 20 to 30 grains, either in one dose just before a paroxysm is expected, or in divided doses during the interval. If it cannot be retained, it may either be combined with an opiate, or be given as an enema. With regard to arsenic, it should be given in the form of Fowler's solution, in doses of ♏ v.-x. three or four times a day. Whichever remedy be selected, it should be continued in smaller doses for at least a week or two after all the symptoms have disappeared. It is also worth while to notice that great importance has been attached to keeping the bowels properly open.

GENERAL DIAGNOSIS OF ERUPTIVE FEVERS.

Cutaneous eruptions constitute one of the most important diagnostic features of the majority of the acute specific fevers; and, by their differences as to date of onset, site of first appearance, and general characters, afford a ready and useful means of distinguishing one fever from another.

If the rash appears within the first four days from the onset of the fever, it is either scarlet fever, measles, epidemic roseola, erysipelas, small-pox, chicken-pox, or glanders. The onset of each is usually well marked; and may be recognised not only by increased heat of the body, but also by the occurrence of rigors, vomiting or convulsions. *If the rash consists of patchy, or more or less diffused redness*, which disappears readily on pressure, and is not attended with any infiltration of the skin, it is either scarlet fever, measles, epidemic roseola, or erysipelas; such specific red rashes may be distinguished from simple roseola, by the very sudden appearance and fugitive character of the latter, the slight febrile disturbance, the absence of sore-throat, the mild constitutional disturbance, and non-infective origin.

	MEASLES.	SCARLET FEVER.	ERYSIPELAS.	EPIDEMIC ROSEOLA.
INITIAL SYMPTOMS.	Those of common catarrh (swelling of face and eyelids, coryza, photophobia, lachrymation and cough). Moderate fever.	High fever, pungent heat of skin, and great frequency of pulse, which set in suddenly and develop rapidly. Initial vomiting is common.	Fever marked in proportion to the eruptive inflammation.	Moderate fever. Slight constitutional disturbance. No coryza. Frequently slight enlargement of cervical glands.
RASH.	Appears on 4th day. Begins on forehead. Brightest on the exposed parts. Dusky red crescentic patches.	Appears on 2nd day. Begins on the neck. Brightest on the covered parts. Bright red points merging into large areas of redness.	Appears in a few hours to 2 or 3 days, and begins generally on the nose or eyelids, and extends thence in one definite direction, generally upwards over the head.	Appears on 1st day. Begins on face. Spots not grouped in crescentic patches, and do not merge into large areas of uniform redness. Considerable irritation of skin.
TONGUE.	Furred.	Red, with prominent papillæ.	Furred, tendency to get dry and brown.	Not characteristic.
THROAT.	Slightly sore.	Soreness generally marked; diffused redness of the fauces and soft palate.	Unaffected.	Slightly sore. No ulceration.
DESQUAMATION.	Slight and branny.	Free and persistent.	Free.	Slight and unimportant.

If the eruption be papular in the first instance it is either small-pox, chicken-pox, or glanders.

Glanders may be distinguished from the above by the history of the case (as a rule it is confined to grooms or stablemen); the rash differs in appearance, and is followed by characteristic rapid ulceration.

	SMALL-POX.	CHICKEN-POX.
INCUBATION.	Constant, lasting 12 days.	Very variable.
ERUPTION.	Appears on 3rd day.	Appears within 24 hours.
	Begins and is most abundant on the face.	Begins and is most abundant on the body.
	Comes out in one crop.	Comes out in successive crops.
	Frequently present on mucous membranes.	*Very rarely* present on mucous membranes.
	Begins as well-marked shotty papule; afterwards a vesicle which is flattened, umbilicated, multilocular; ultimately a pustule which is matured by the 9th day.	Papules imperfect, and become vesicular in the course of a few hours. Vesicles hemispherical one-chambered, not umbilicated, and do not become pustular.
INITIAL SYMPTOMS.	Pain in the back, vomiting, and high fever.	Very slight indeed, and often not observed at all.
DURATION.	3 to 5 weeks.	Average 7 to 10 days.

The only other eruptive fevers are typhus and typhoid, which may be thus contrasted:—

TYPHUS FEVER.	TYPHOID FEVER.
May occur at any age.	Rare after middle age.
Invasion more or less abrupt.	Invasion gradual.
Face dusky; expression heavy; and eyes injected and suffused.	Face pale, with a circumscribed flush on each cheek.
No special abdominal symptoms, or diarrhœa.	Typical abdominal symptoms, and diarrhœa as a rule.
Prostration and low nervous symptoms appear early.	Low nervous symptoms, if present, appear much later.
Special eruption which appears on the 4th or 5th day; first on the back of wrist, and epigastrium; comes out in one crop; and does not disappear on pressure.	Special eruption which appears from 7th to 12th day; first on the abdomen or back; comes out in successive crops, and disappears on pressure.
Special course of temperature. (Refer).	Special course of temperature. (Refer).
Duration shorter than that of typhoid.	Duration longer than that of typhus.

SECTION III.

GENERAL DISEASES. (B.)

RHEUMATISM.

THE terms *rheumatism* and *rheumatic* have been very loosely employed to denote an inflammation or painful affection of *any part* arising from exposure to cold or damp; but they will here be exclusively applied to certain inflammatory and painful conditions of the joints, or of the muscles and fibrous tissues in connection with them.

1. ACUTE ARTICULAR RHEUMATISM—RHEUMATIC FEVER.

The conditions which lead to articular rheumatism are two in number:—(1) A constitutional predisposition which may be hereditary or acquired; and (2) an exciting cause, which is generally exposure to cold or damp. As to the precise nature of the pathological disturbance we have yet much to learn; but there is strong evidence pointing to a disturbed metabolism, which has its chief seat in the liver, leading to the formation of some abnormal chemical product set free in the blood. The disease is most common between the ages of 16 and 26.

MORBID ANATOMY.—The changes are those of an acute superficial inflammation of the lining membrane of certain joints. The interior of the joint becomes congested, and loses its natural polish. An effusion into the joint-cavity usually follows, which consists of clear or slightly turbid serosity, with flakes of lymph in suspension. Suppuration is very rare. Similar changes are apt to affect the sheaths of tendons, and the synovial bursæ. There is often a little œdema of the tissues around the joint. Small subcutaneous fibroid nodules are occasionally met with, especially in children, in the neighbourhood of the joints, and over bony prominences elsewhere.

SYMPTOMS.—The actual attack is characterised by inflammation of the joints, fever, profuse sweating, and anæmia; and is usually preceded for a few days by wandering pains in the joints and muscles accompanied with more or less malaise.

Local Signs.—The joints usually affected are of middle size, viz., the knees, ankles, elbows, and wrists. The affected joint is extremely painful, (the pain being intensified by the least movement), tender, swollen, and hot. The swelling is mainly due to fluid effusion within the joint, but partly to œdema of the tissues around it. The skin is slightly reddened or unaltered. The number of joints attacked at one time varies greatly; but the inflammation has a great tendency to migrate suddenly from one joint to another. Similar changes may be observed in connection with the sheaths of certain tendons, or synovial bursæ.

General Symptoms.—The amount of *fever* varies much, but it rarely exceeds 104° in uncomplicated cases. It is characterised by very variable daily remissions, and by considerable irregularities in its course. It subsides by *lysis*, concurrently with the articular inflammation. *Profuse perspiration* with a sour smell, of very acid reaction, and often accompanied with sweat rashes (sudamina and miliaria), is an almost invariable symptom. The very rapid development of *anæmia* requires special notice, and is in direct proportion to the severity, and the duration of the disease. The urine is scanty, high-coloured, very acid, and deposits urates in abundance.

The bowels are usually constipated. Delirium is rarely met with except under one of the following conditions:—(*a*) some inflammatory complication—more often, pericarditis; (*b*) previous alcoholism; and (*c*) as a premonitory symptom of hyperpyrexia.

Endocarditis, leading to valvular disease, is by far the most frequent and most important complication of acute rheumatism. Pericarditis also occurs not infrequently; and in such cases usually co-exists with endocarditis. Among the rarer complications are pneumonia, pleurisy, meningitis, and a peculiar cerebral affection coming on suddenly with delirium, and hyperpyrexia, followed by coma, and rapidly fatal, of which the precise nature is unknown. Rheumatic subjects are also especially liable to tonsillitis, and erythematous skin eruptions.

The *duration* depends entirely upon the severity of the case, and the kind of treatment adopted, varying from one to six weeks. There is great liability both to relapse and recurrence, especially in feeble subjects.

The *diagnosis* from gout will be given when describing that disease.

The *prognosis* is always favourable in the absence of complications.

TREATMENT.—Under all circumstances, rest in bed should be insisted upon, and the joints should be wrapped in cotton wool. The diet should be liberal though plain and light; alcoholic stimulants are rarely required.

As to medicinal remedies many have been from time to time recommended. Of these, the salts of soda and potash—given in large doses, combined or not with quinine or opium—tincture of the perchloride of iron, salicine, salicylic acid, and the alkaline salicylates, are the most important. Treatment by the salicylates of ammonia, soda, or potash, is generally recognised as superior to any other method. The salicylate should be given in doses of gr. x. to xx., repeated every two or three hours until the pains subside, and afterwards less frequently until the temperature has returned to the normal level. It is readily taken, and unpleasant symptoms rarely occur. Occasionally, however, there may be some headache, buzzing in the ears, deafness, and slight mental confusion, but these symptoms quickly subside on suspending the use of the drug. I now rarely see an uncomplicated case (if treated early) last longer than a week or ten days, when treated in this way. The remedy should be continued in smaller doses for at least a week or ten days after the cessation of acute symptoms. The secretions must be attended to; and great comfort is sometimes derived from sponging the body with tepid water, of course avoiding all unnecessary exposure.

The local application of poppy fomentations, or light poultices, sometimes gives relief to the pain in the joints. Blisters cannot be recommended, and when employed, should be used with great caution. Hyperpyrexia calls for prompt antipyretic treatment, by repeated cold wet-packs, or better still, when possible, by immersion of the patient in a bath of about 85°, afterwards cooled down to about 75°. Such baths may be repeated as occasion requires.

Care should be taken during convalescence, to avoid exposure to cold or damp. Quinine in combination with iodide of potassium is a useful tonic at this stage.

2. CHRONIC ARTICULAR RHEUMATISM.

Chronic rheumatism is most common in the latter half of life. Not infrequently it forms a sequel of the acute variety. But more often it is chronic from the beginning. The predisposing and exciting causes are also probably identical with those of acute rheumatism.

It is characterised by pain, tenderness, and stiffness in the affected joints, not infrequently accompanied with crackling or grating sounds on forced passive movements. The amount of structural change in the joints varies widely. It may not be obviously altered on external observation; but on the other hand there may be enlargement, thickening, or (in severe and long standing cases) marked deformity. The latter cases merge imperceptibly with rheumatoid arthritis; and in the present state of our knowledge they cannot be distinguished.

Besides these local symptoms, there is usually anæmia, with feeble digestion, and more or less general debility. The subjects of the disease are also remarkably sensitive to cold, damp, and changes in the weather. Cardiac complications may sometimes occur, and it is not improbable that some valvular diseases of the heart which appear to have arisen spontaneously, may in reality be traceable to chronic rheumatism which has been overlooked.

TREATMENT.—Flannel should be worn, and all exposure to cold or damp avoided. The skin should be maintained in a healthy active condition by baths (hot air, vapour, or water) and frictions. The diet should be light and bland; and gentle exercise in the open air is desirable.

Locally, frictions, counter-irritation with iodine stimulating liniments, or strapping the joint with simple or medicated plasters, may be employed with advantage, according to circumstances.

Medicinally, tonics, cod-liver oil, iodide of potassium, guaiacum, and liq. arsenic. hydrochlor., are among the most useful remedies. Salicine and the alkaline salicylates are of little or no service in chronic rheumatism.

3. Gonorrhœal Rheumatism.

This is sometimes described as a variety of articular rheumatism. It arises in the course of, or as a sequel to an attack of gonorrhœa; and it is mainly characterised by very considerable effusion into the affected joint (most frequently the knee); a tendency to become intractable and chronic; anatomical changes in the joint-structures which are apt to end in more or less anchylosis; and a great liability to recurrence. Otherwise it resembles acute articular rheumatism, and calls for essentially similar treatment. Co-existing gonorrhœa of course requires attention.

4. Rheumatoid Arthritis—Arthritis Deformans.

In deference to prevailing opinion, rheumatoid arthritis is here described separately from other forms of articular rheumatism. If there be any real distinction between them, it lies in some special characters belonging to their remote pathological cause, and of this we do not as yet possess any precise knowledge. The most obvious distinguishing feature is the amount of local swelling and deformity (which would seem to be solely related to the severity of the inflammatory change) and its extension beyond the limits of the synovial membrane to the cartilage and the structures around the affected joints.

Rheumatoid arthritis is more common in the latter half of life, but it may occur at all ages. It is also undoubtedly hereditary, and is encouraged by depressing influences of any kind. As to its general course it is chronic, but it rather consists of a series of acute or subacute attacks, recurring with increasingly short intervals, each leaving more permanent structural change in the joints, till the disease becomes practically persistent.

Morbid Anatomy.—The articular cartilages are affected from the outset. At first they present a velvety appearance, but afterwards become worn down, exposing a bony surface, of the smoothness and hardness of ivory (*eburnation*). The synovial fringes are thickened and form bulb-like clusters at the circumference of the joint, which are afterwards converted into cartilage or bone. Similar thickening enchondrosis and ossification occur in connection with the periosteum, tendons, and ligaments about the joints. The synovia is not materially increased.

Symptoms.—The onset is marked by more or less severe pain in the joint (intensified on movement) tenderness on pressure, with some slight febrile disturbance. The joints swell, but as the swelling increases the pain often subsides. Such attacks frequently recur, affecting a gradually increasing number of joints, which sooner or later become permanently thickened, characteristically deformed, and the muscles in connection with them are generally much wasted. The disease usually begins in the fingers, but it may attack any or every joint. The deformity of the hands is very characteristic, the fingers overlap each other, are directed to the ulnar side, the first and third phalanges being flexed and the second extended. Sometimes (especially in old people) one joint alone is affected, more often the hip or the shoulder; and the deformity which follows may in such cases lead to some diagnostic confusion with dislocation or intracapsular fracture.

Diagnosis.—(Refer to "Diagnosis of Gout.")

The *prognosis* is unfavourable in proportion to the chronicity of the disorder.

Treatment.—The course of rheumatoid arthritis is generally very tedious and unsatisfactory. A light nutritious diet is called for; and the addition of some light dry wine is often of service. Exercise in the open air should be resorted to daily, where possible. Above all it is of the greatest importance to maintain the action of the skin by baths, frictions and otherwise. As internal remedies—general tonics, arsenic, guaiacum, iodide of potassium, actæa racemosa, are chiefly recommended. With regard to local treatment, rest and mild counter-irritation are indicated in the earlier and more acute stage; while later on, passive motion friction or strapping are often beneficial.

5. Muscular Rheumatism—Myalgia.

Under this term are included certain painful affections of the muscles, and the fibrous structures in connection with them. Nothing is known about the morbid anatomy of muscular rheumatism. Special terms are applied to certain varieties. Thus, *pleurodynia*, when the intercostal and thoracic muscles are affected; *lumbago*, when the muscles of the loin are affected; *torticollis*, when the sterno-mastoid and other muscles of the neck are involved. The last should be distinguished from spasmodic torticollis, which is due to other causes. The pain is felt when the muscle is thrown into contraction by movement, and is accompanied with more or less tenderness on pressure. The constitutional symptoms, if present, are those of mild fever.

Treatment.—A hot bath, followed by a brisk purge, is useful at the outset; and among internal remedies, salines, chloride of ammonium, alkaline salicylates, and iodide of potassium, are the best. Locally, pain may be relieved by hot fomentations or poultices, anodyne liniments, and firm strapping (when possible). Warm clothing is also of great importance.

GOUT—PODAGRA.

Gout, though usually *hereditary*, is sometimes *acquired*. As a rule it first appears in the latter half of life; but it appears earlier when inherited, than when acquired.

The most prominent pathological feature of gout, is an *accumulation of uric acid in the system*. This condition has been variously attributed to defective elimination, functional disorder of the liver, special degenerative change in the tissues, and perverted innervation. But whatever may be the details of its causation, we may safely assume the existence of a gouty predisposition (inherited or acquired) which is characterised by weak and faulty metabolism; and that an actual attack may be induced in one so predisposed, by anything which over-taxes or reduces metabolic capacity. Thus, luxurious living with eating and drinking in excess overtax metabolism; while alcohol, want of fresh air, deficient exercise, and nervous depression reduce it. In practice, these causes are often combined.

Morbid Anatomy.—In the first place a deposition of crystals of urate of soda occurs in the substance of the articular cartilages, synovial fringes, and fibrous structures of the joints. The tendons, bursæ, periosteum, internal organs, and fibrous tissue elsewhere (especially of the lobules of the ear) may become the seat of similar deposits, which, when large enough to be felt during life, are called "tophi," or from their white chalk-like appearance "chalk stones." Ordinary inflammatory changes now supervene where the deposit has formed. The proximal joint of the great toe is generally first attacked, but as the disease progresses, several joints are involved which tend to become stiff and anchylosed. The skin overlying the concretions may ulcerate, and lead to the formation of abscesses, which discharge pus and "chalky" matter.

Symptoms.—When gout is limited to the joints it is said to be *regular*, and in this form is *acute* at first, but afterwards becomes *chronic*. When, however, it is characterised mainly by visceral disturbance, it is termed *irregular* or *retrocedent*.

An attack of *acute regular gout* is generally preceded for a variable time by digestive troubles, acidity, heartburn, nervous irritability, and scanty acid urine; but it may occur suddenly without previous warning. The onset is usually during the night, in the course of which the patient awakes with violent pain in the proximal joint of one great toe. This is soon followed by heat, redness, and swelling of the skin over the joint, which then becomes smooth, shining, and marked by blue dilated veins. These changes are accompanied with more or less febrile disturbance, irregular sour-smelling sweats, anorexia, constipation, and extreme restlessness. The urine is scanty, high coloured, with a thick deposit of urates, *the total amount of uric acid being less than normal*. The symptoms usually abate during the daytime, and return each night, but with diminishing severity. At the end of about a week (in first attacks) the pain, redness, and swelling of the joint subside, and after desquamation of the cuticle around, it returns to its normal condition.

These attacks are very liable to recur, tending to become less acute and of longer duration, to involve a larger number of joints, to attack other parts, such as the heel or the instep, and to return at shorter intervals as the case proceeds, until it finally passes into the *chronic* form.

There is now less fever, but the pain though less acute is almost constant. Several joints are affected, around which "chalk-stones" are apt to form, causing enlargements, deformities, and stiffness. Nodular deposits of a similar kind may occur in the lobule of the ear, and elsewhere. The health is greatly impaired, and the patient complains of acid eructations, flatulence, loss of appetite, irregular action of the bowels, circulatory disturbance, and great irritability.

Occasionally, in the place of the usual articular changes, the internal organs may be attacked (*retrocedent*, or *irregular gout*). Thus it may affect (1) *the brain*, causing intense headache, delirium, or convulsions; (2) *the stomach*, causing gastralgia, nausea, vomiting, &c.; (3) *the heart*, giving rise to irregular or intermittent cardiac action, palpitation and attacks of syncope or angina pectoris; or (4) *the lungs*, causing bronchitis and asthma.

The subjects of gout are especially liable to obstinate eruptions of the skin; to a form of chronic Bright's disease (granular kidney); and to arterial degeneration, a common predisposing cause of apoplexy.

For the purposes of *diagnosis* the following table* may be of service.

PROGNOSIS.—Gout usually runs a chronic course with little direct danger to life. Death, when it occurs, is due either to some complication or intercurrent disease.

TREATMENT.—Plain living, a restricted amount of animal food, avoidance of alcoholic stimulants, plenty of

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* Adapted from Roberts' *Handbook of Medicine*.

ACUTE GOUT.	ACUTE RHEUMATISM.	RHEUMATOID ARTHRITIS.
1. Rare in early life.	1. Common in early life.	1. May occur at all ages.
2. Smaller joints chiefly affected, especially the great toe. Not erratic. Joint very œdematous, red, smooth, shining, with enlarged veins. Cuticle finally desquamates.	2. Medium sized joints chiefly affected. Very erratic. Swelling of joint mainly due to effusion within it. Skin only slightly altered. No enlargement of veins. No desquamation.	2. Smaller joints more often first affected. Not big toe specially. Not erratic. Considerable swelling and thickening of all joint-structures, followed by deformity.
3. Urate of soda deposited in the joint.	3. No deposit of urate of soda.	3. No deposit of urate of soda.
4. Attacks of short duration, and with great tendency to periodical recurrence.	4. Attacks of longer duration, with less liability to recur. No periodicity.	4. Special tendency to become chronic.
5. *Complications.*—Severe functional disturbance of brain, stomach, heart, or lungs.	5. *Complications.*—Endocarditis, pericarditis, and inflammation of lung or pleura.	5. No special complications.
6. *Urine.*—Scanty, acid; deficient in uric acid at first; afterwards in excess. Albuminuria common.	6. *Urine.*—Febrile; sometimes slight albuminuria.	6. Nothing special in the urine.

exercise in the open air, encouragement of the action of the skin and bowels, and as much freedom as possible from worry and anxiety will suffice to ward off, if not altogether to prevent, an attack of acute gout in the early stages of the disease. In the more chronic forms, however, the patient is often so crippled, and his general health so broken, that a liberal diet with stimulants may become necessary.

During the attack the joint must be rested, and evaporating lotions or anodyne applications may be employed to relieve the local pain. Internally, *after* the bowels have been freely opened, it is well to administer full doses of salicine, in combination with an alkaline bicarbonate and colchicum, (vini ♏x.-xv.) three or four times a day, until the attack subsides. The salts of lithia or potash are preferable to those of soda. Generally speaking the emunctories should be encouraged to act pretty freely by systematic bathing, followed by friction of the skin, drinking of mild alkaline diluents in abundance, and the occasional use of an aperient. In the chronic forms of gout, alkalies freely diluted, guaiacum, and iodide of potassium are of value, but attention to dietetic and hygienic matters is of still greater importance.

RICKETS—RACHITIS.

Rickets is a form of general malnutrition, which is specially characterised by peculiar changes in the bones. It is essentially a disease of childhood, and seldom occurs before the 6th or after the 18th month. It is proximately induced by improper food, and especially in relation to a deficiency of fatty and proteid constituents. Anything however which interferes with development and nutrition, such as inherited weakness or faulty sanitation, favours its occurrence.

Morbid Anatomy.—The changes in the bones consist of an excessive proliferation of the deeper layers of the cartilages and periosteum, together with delay in the process of ossification. The bones are consequently increased in thickness, especially at their ends; and, moreover, they become softened, so that they are peculiarly apt to bend or break. After awhile ossification is once more resumed, and the new-formed bone is extremely hard and dense. The liver, spleen, and lymphatic glands are also often enlarged. As yet we have little or no precise knowledge to account for these remarkable changes.

Symptoms.—Three signs stand out as especially characteristic of the onset of rickets:—(1) Profuse sweating of the head, face, and upper part of the chest, increased during sleep, or on the slightest exertion when awake; (2) a strong desire to be cool at night by kicking off the bed-clothes; and (3) a peculiar tenderness of the body when moved or pressed, which indicates the beginning of morbid changes in the bones. Notwithstanding that rachitic children are sometimes unusually fat, the flesh is soft and flabby, the skin is pale, the bowels act irregularly, the motions are offensive, and the appetite is often ravenous. The cutting of the teeth is delayed, and if the child has already begun to walk, it now ceases to do so, sits "all in a heap," suggesting to the anxious mother that the back is "growing out." The child is weak, languid, peevish, and restless.

Before long, the ends of the long bones become enlarged. This is most easily observed at the junction of the ribs with their cartilages, but the wrists, ankles, and other joints are very often affected. The bones also become softer and more or less thickened throughout. If the disease is unchecked, deformities follow. Thus, the limbs may become bowed, the ribs may be drawn in anteriorly by the action of the diaphragm, thrusting the sternum forward (*pigeon-breast*); the spine may be variously curved; and, in extreme cases, the antero-posterior diameter of the pelvis may be shortened by pressure of the sacrum forwards into the pelvic cavity. The head is large, out of all proportion to the face; the forehead is high, square, and projecting; and the closure of the fontanelles is delayed. Owing to the resistance offered by the solid organs in the upper part of the abdomen, the lower ribs are not drawn in like those above, and this gives rise to a relative enlargement of the lower part of the chest. The abdomen is large and distended.

As the disease progresses, the child becomes shrunken and old-looking, the digestive functions are increasingly disturbed, the bowels are most irregular, and the nervous system acquires a peculiar excitability and liability to grave disturbance from slight causes. The urine is increased in amount, and often turbid from earthy phosphates. Inflammatory and nervous complications are common enough, and often prove fatal, but as a rule the affection is remarkably amenable to treatment, if not too long delayed. During convalescence, ossification of the softened bones takes place, so that they once more become hard and firm; but stunted growth, and persistence of any deformity which may already have taken place, constitute permanent indications of the past disease.

The *complications* of rickets are both numerous and important. Bronchial catarrh, diarrhœa, laryngismus stridulus, convulsive affections generally, and chronic hydrocephalus, are those most commonly met with.

The *diagnosis* in well-marked cases is attended with no difficulty whatever. In the milder forms, most reliance may be placed on the initial symptoms (as already described), the delayed dentition, and the lateness in walking and talking. It is distinguished from *mollities ossium* in that the latter is a disease of adults, and is never followed by re-ossification.

TREATMENT.—The diet claims foremost attention in the treatment of rickets, and many cases may be cured entirely by qualitative adjustment of the food. Fresh cow's milk, diluted with barley-water according to the child's age and digestive capacity, with added proteid and fat in the shape of meat-juice and cream, is the food which is most generally suitable. Starchy food should be altogether avoided by rachitic infants under one year old. With older children, on the other hand, the milk diet may be supplemented by animal broths and the yolk of egg. Digestive disorder may call for modification of the above diet; but the principle of providing a sufficiency of proteid and fatty food must not be lost sight of. Abundance of fresh air, sunlight, and cleanliness of surroundings rank only next in importance to diet. Drugs are useful adjuncts in many cases, especially cod-liver oil, malt extract, preparations of iron, and quinine. Lime salts have also been recommended; and I have sometimes seen benefit from a course of chloride of calcium (dose, gr. i.-iij.). Where the bowels are irregular and the stools offensive, a mild aperient is necessary at first, followed by a few grains of some alkaline bicarbonate or liquor potassæ, in combination with nux vomica or some vegetable bitter which may be repeated three or four times a day for a while. Obstinate constipation is best treated by warm clothing, frictions of the abdomen, and occasional enemata; should any aperient be necessary, a dose of castor oil may be given; or better still, one or two drops on sugar, once or twice a day, of a solution of podophyllin in spirits of wine (gr. j. ad ʒ j.). As to the deformities, children should be prevented from getting about too early; and in recent cases where the bending of the limbs is marked, gentle extension with splints may be employed. Complications must be treated as they arise.

SYPHILIS.

Syphilis is a specific disease, which may be either acquired or inherited. In the former case it can only be communicated by inoculation, which occurs for the most part during sexual intercourse with an infected person. In the latter case it is transmitted from the parent to the offspring *in utero*. One attack of the *primary* disease would appear to confer protection against its repetition.

Primary Syphilis.—About 28 days after inoculation a "primary sore" appears, which is characterised by a superficial ulcer with an elevated margin, and an indurated base. After about two months it gradually cicatrises. The lymphatic glands in connection with the affected part begin to enlarge shortly after the commencement of the "sore," but they have little or no tendency to suppurate. After the disappearance of the sore they usually soon regain their normal size, or nearly so; but sometimes they remain more or less enlarged for months or even years.

Secondary Syphilis.—From six weeks to three months after the first appearance of the primary sore, some slight febrile disturbance, debility, and anæmia, usher in a group of so-called *secondary symptoms*, which present considerable variety. The most important are as follows:—

1. *Cutaneous eruptions*, which may be macular, papular, vesicular, scaly, or pustular. They are mainly characterised by (*a*) a coppery colour, (*b*) a brownish stain left for a considerable time after their disappearance; (*c*) the occurrence of various types of eruption at the same time; *(d)* a tendency to circular grouping; (*e*) an absence of itching; and *(f)* by an aptitude to affect parts such as the forehead, soles of the feet, and palms of the hands, which are rarely affected by non-specific eruptions. The hair is also liable to become dry and fall off. 2. *Lesions of mucous membranes*, including (*a*) superficial ulcers of the tonsils, soft palate, &c.; and (*b*) mucous tubercles of the orifices of the mouth, nose, rectum, or elsewhere. 3. *Lesions of the eye*, including (*a*) iritis; and (*b*) choroido-retinitis. 4. *Pains in the joints and bones*, due to slight periostitis.

Tertiary Syphilis.—Unless the further progress of the disease be checked by appropriate treatment, a fresh series of lesions may occur (*tertiary syphilis*), after an interval which varies from one to several years. There is no sharp distinction between secondary symptoms on the one hand and tertiary symptoms on the other; but the latter are mainly characterised by their late appearance, irregular distribution, the tendency to involve a considerable thickness of tissue with liability to deep ulceration, and frequent implication of viscera and other parts by the formation of gummata. The most important are as follows:—

1. *Cutaneous lesions*—rupia, tubercular formations, and ulceration. 2. *Lesions of mucous membranes*—destructive ulceration of the soft palate, pharynx, or rectum. 3. *Gummatous tumours*, composed of a fibro-nuclear growth infiltrating the sub-cutaneous, or sub-mucous tissues, the muscles or internal organs. 4. *Periostitis and necrosis of the bones*, most frequently affecting the tibia or the bones of the skull.

Sooner or later, if the progress of the disease be still unchecked, the so-called syphilitic cachexia sets in, and the patient may finally die either from exhaustion or from some intercurrent affection.

Congenital Syphilis.—Symptoms usually first appear at the end of the third to the sixth week after birth, in the form of cutaneous eruptions resembling those of the secondary period of acquired syphilis, and of most frequent occurrence on the nates, abdomen, palms and soles. Congestion and swelling of the nasal mucous membrane, with increased secretion, also constitute common initial symptoms (*snuffles*). Mucous tubercles about the mouth or anus, and interstitial inflammation of the cornea are frequently met with. The temporary incisor teeth are cut early and as a rule with little difficulty; but they soon decay, and the permanent teeth which replace them are ill-formed, short, peg-shaped, and present a central notch. If the disease proceed unchecked, symptoms of the tertiary type may occur. The child's appearance is cachectic, the features pinched, the skin dry and shrivelled, and it gradually sinks. On the other hand appropriate treatment by mercurials will speedily restore the child, in most cases, to a condition of comparative health, if begun sufficiently early.

Treatment.—The only effectual remedy for syphilis is mercury. It may be given either (*a*) by *inunction* with mercurial ointment; (*b*) by *fumigation* with the vapour of calomel; or (*c*) by *internal administration* of some mercurial preparation. For infants the method of inunction is the most convenient. For adults, in all ordinary cases, nothing answers better than gr. j. doses of mercury and chalk three or four times a day in the form of a pill made up with a little ext. gentian. The treatment should never be pushed to the extent of salivation, and should be continued for at least one year after the disappearance of symptoms, in smaller doses given less frequently. When more rapid effects are desired, the employment of fumigation, or of blue pill (gr. ij.-iij. *ter die*) with a little opium is preferable. Iodide of potassium is of special service in tertiary syphilitic affections of the internal organs, particularly of the nervous system. In such cases the dose should be free, and be rapidly increased. Above all it should be remembered that syphilis is a debilitating disease, and the importance of all treatment which tends to the improvement of the general health cannot be over-rated.

Further details must be sought in surgical text-books.

SCORBUTUS—SCURVY.

Scurvy is a disorder which primarily depends on a morbid condition of the blood, arising from a deficient supply of certain substances necessary for proper nutrition, more particularly of the salts of potash with vegetable acids. As powerful auxiliary causes, cold, damp, hardship, and depressing influences generally, must be mentioned.

Morbid Anatomy.—The blood is profoundly altered, being liquid and dark coloured. Ecchymoses are seen on the surface of the skin, serous and mucous membranes. Firm coagulated extravasations are also met with beneath the skin, and between the muscles. Blood-stained effusions may occupy the serous cavities.

Symptoms and Course.—A sense of lassitude, debility, and disinclination for exertion of any kind, often accompanied with darting pains in the limbs and joints, usually precedes the characteristic changes in the gums, which become swollen, soft, and spongy, and bleed on the slightest touch. The teeth are tender and loosened. Subcutaneous hæmorrhages (*petechiæ*) occur, and are most abundant over the lower limbs; while not infrequently deep-seated extravasation gives rise to brawny indurations about the hams and elsewhere, rendering all movements of the limbs difficult and painful. Hæmorrhages from the various mucous surfaces are also liable to occur. The general condition is characterised by debility and prostration, the skin is sallow and harsh, the breath is offensive, the appetite fails, and the bowels are generally constipated. There is no fever in uncomplicated cases. If not relieved, the prostration increases, the pulse becomes very feeble, the least exertion is attended with dyspnœa and palpitation, and the integument is often œdematous and sometimes the seat of obstinate ulceration. Prompt and appropriate treatment is usually followed by recovery; otherwise the case drags on from bad to worse, till death occurs from exhaustion or some complication.

Treatment.—It is important without delay to restore those substances to the system, to the want of which the disease is primarily due, viz., the vegetable salts of potash. For this purpose, fresh vegetables and green stuff should be freely supplied. Lime or lemon juice (℥j.-℥ij. in a tumbler full of water, and sweetened, taken thrice daily) is also of great value.

The diet should be light, nutritious, and liquid until the condition of the gums is improved, and the digestion is stronger. Stimulants are occasionally required.

In the absence of fresh vegetables or lime juice, citrate of potash may be given with advantage.

PURPURA.

Little is at present known of purpura beyond the fact that it is a morbid condition which is characterised by hæmorrhages under the skin, and often also from other parts. It is met with under a variety of circumstances —from general personal neglect and bad feeding; in acute fevers (hæmorrhagic varieties); in the course of certain chronic diseases (*e.g.*, leukæmia and Bright's disease); in association with some skin eruptions (*e.g.*, purpura urticans); and sometimes in those who have been otherwise healthy (*primary purpura*).

It is probable that in all cases the hæmorrhages are proximately due to one or both of two conditions, namely, malnutrition of the minute blood-vessels, and some alteration in the constitution of the blood; but as to the way in which these are brought about, we have no precise knowledge.

Symptoms.—It is only possible to consider the symptomatology of purpura in those cases which are unaccompanied with any other obvious disease. Under these circumstances the skin becomes studded with hæmorrhagic extravasations, which vary in size from minute petechiæ, to large and irregular ecchymoses, and present the usual changes of colour which characterise bruises. They are most abundant on the legs and lower part of the trunk, and generally appear in successive crops. There is especial liability to hæmorrhage from the various mucous surfaces, namely, epistaxis, hæmoptysis, hæmatemesis, hæmaturia, and menorrhagia. The constitutional symptoms are mainly characterised by debility and depression, which are proportionate to the severity of the case, and the loss of blood. Recovery generally takes place in about a fortnight. In those cases where purpuric hæmorrhage complicates some other disease, it obviously increases the gravity of the prognosis; and the hæmorrhagic forms of the acute specific fevers are almost always fatal.

Scurvy is distinguished from purpura by the marked cachexia, the affection of the gums, and its dependence upon a want of fresh vegetables. Hæmorrhages from the mucous surfaces are also more common and abundant in purpura than in scurvy.

The *treatment* is necessarily of a general kind, and attention to the hygienic surroundings of the patient, a liberal supply of good food, with warmth and rest, should constitute its leading features. Medicinally, iron and quinine with sulphuric acid are of most service. Internal hæmorrhages are best checked by the administration of turpentine in doses of ♏ x.-xx. with mist. amygdalæ, frequently repeated.

SCROFULOSIS—STRUMA.

Scrofulosis or Struma is a constitutional condition which is manifested by a peculiar tendency to inflammation in various parts of the body, arising from trivial causes. The inflammatory process, moreover, is characterised by extreme chronicity, profuse cell-proliferation, and liability to consecutive caseous degeneration. Inflammation of this kind is especially apt to invade the lymphatic glands, skin, mucous membranes, joints and bones. Great stress is usually laid upon the frequency of inflammation of the lymphatic glands, as being peculiarly characteristic of the scrofulous habit; and undoubtedly glandular enlargements often constitute the first obvious outward indication of scrofulous mischief. It must be remembered, however, that the glands do not enlarge spontaneously, but only under the influence of irritation set up by some antecedent lesion of an inflammatory nature, to which the scrofulous habit renders them peculiarly susceptible. Thus, enlargement of the cervical glands commonly arise from preceding inflammation of the tonsils; enlargement of the bronchial glands from bronchial inflammation; and enlargement of the mesenteric glands from intestinal inflammation.

The liability of scrofulous subjects to develop tuberculosis has long been known, and discussion as to the exact nature of the relationship between these two conditions has led to endless confusion of the whole subject. The recent investigations of Koch, which clearly stamp tuberculosis as a specific infective disease, go a long way towards permanently clearing up the difficulty. The nature of the relation according to this view is simple enough, and amounts to this—that chronic scrofulous inflammation affords peculiarly favourable conditions for the growth and development of the tubercular virus, now conclusively proved to consist of specific micro-organisms. There is in other words no *necessary* relation between them at all; and so long as the system is free from the specific tubercular virus, scrofulosis will never lead to tuberculosis.

Heredity is the most important predisposing cause of scrofula; but it may be also undoubtedly acquired by anything which tends seriously to lower the general health, and to impair development. It is essentially a disease of childhood and early life.

Symptoms.—Children who inherit a scrofulous taint are often characterised by a short stumpy figure, thick opaque skin, round face, ugly features, coarse hair, thick upper lip, broad nose, dull temperament, and backward intellect. The bones are thick, the teeth decay early, and the belly is tumid.

With regard to the special manifestations of struma there is nothing peculiar beyond what has been stated when defining the nature of the disease. As in syphilis, affections of the skin and glands often constitute the earliest lesions; subsequently the mucous membranes and cellular tissues are apt to be attacked; while still later in bad cases, the bones and joints are prone to suffer. At the same time, departure from this sequence of events is of frequent occurrence. Among the more frequent affections thus met with, are enlargement and suppuration of lymphatic glands, pustular eruptions, tinea tarsi, otorrhœa, ozæna, vulvitis, cold abscess, synovitis, and ostitis with caries. Should the morbid diathesis remain unchecked by treatment, secondary diseases such as tuberculosis, pulmonary phthisis, and albuminoid degeneration of various organs may arise.

The *treatment* of scrofula is primarily hygienic. Fresh air, exercise, baths, good food, (especially animal food), are chiefly required. Medicinally, bitter tonics, cod-liver oil and preparations of iodine are indicated.

TUBERCULOSIS.

In the light of recent knowledge, tuberculosis must be regarded as a specific infective disease which only arises on the introduction within the body of a specific bacterial organism, which alone constitutes the tubercular virus. The organism in question is a slender bacillus, about one-third of the diameter of a red blood-cell in length. It is peculiar for the slowness of the multiplication and diffusion through the body, as compared with other analogous organisms. For a long time its development may be confined to a particular locality, *e.g.*, the lungs; and death may occur as the result cf the local disease thus set up, before the system as a whole becomes involved. Under other circumstances, its development may be general from the outset.

But in order that the growth and development of the tubercular virus may take place, a predisposition on the part of the individual attacked is necessary, without which tubercular disease does not occur. This predisposition may be either general or local. The scrofulous diathesis perhaps constitutes the most important form ot general predisposition. But a constitutional predisposition to tuberculosis, other than scrofula, may be either inherited or acquired; and in the former case is often recognisable by a special build and appearance of the body. If the term "tubercular diathesis" is to be retained at all, it may be fairly applied to this condition. As to local predisposition, it would appear that any antecedent disease might act in this way, in so far as it lowered the standard of nutrition of a part.

The characteristic anatomical feature of tuberculosis consists in the formation of so-called "tubercles" in the affected part. Two kinds of tubercles have been described, namely *grey* and *yellow*. The latter, however, is now known to depend upon caseation, not only of grey tubercles, but of other inflammatory products. True tubercles consist of pearly-looking, greyish, non-vascular nodules about the size of millet-seeds when isolated; but larger nodules may result from fusion of adjacent tubercles. Tubercles may be found in the lungs, intestines, lymphatic glands, serous membranes, spleen, and other viscera. Microscopically, a typical tubercle consists of a centrally situated many-nucleated giant-cell, surrounded by a zone of largish cells of an epithelioid type with oval nuclei; outside which is another zone of small lymphoid cells; the whole being imbedded in a reticulum of doubtful nature. While this is the usual structure of a fully formed tubercle, in its early stage before it has begun to degenerate, it cannot be regarded as absolutely characteristic. It is the circumstances under which tubercles occur, rather than their anatomical appearances which are specific.

Bacilli, such as already described, are found as a rule both inside and outside the nodule, and are arranged either singly or in groups. Later on, the centre of the tubercle becomes opaque, yellowish and granular from degenerative changes. The further progress varies. 1. Re-absorption (?) may occur. 2. The nodule may caseate, soften, and become converted into a puriform liquid, forming an ulcer when superficial, or a pseudo-abscess when situated more deeply. 3. It may caseate and calcify, becoming quiescent and inert. 4. It may undergo fibroid organisation.

A tubercle must be considered as a local inflammatory new formation of lymphoid tissue, primarily set up by tubercle-bacilli which have found their way to the part. Its cellular elements are probably derived from multiplication and metamorphosis of leucocytes, or connective-tissue cells; but the share (if any) which is taken by each, remains to be ascertained. More or less diffuse inflammation of the surrounding tissue always occurs. The process extends by the formation of fresh nodules in the immediate neighbourhood of the part just affected, or the virus may be carried to distant parts by the lymphatics or blood-vessels.

Symptoms.—As above remarked, certain children other than strumous inherit a special susceptibility to the development of tubercle. They are often characterised by a tall thin figure, thin soft delicate skin, oval face, regular features, silky hair, large bright eyes with large eyelashes, excitable temperament, and precocious intellect. The symptoms of tuberculosis necessarily vary according to the organ affected, and will be described among the local diseases. There is a form, however, in which the tubercular formations are not confined entirely or even principally to one part, but are scattered throughout the body. Acute general tuberculosis gives rise to symptoms which are very like those of typhoid fever, but the absence of any eruption, typical diarrhœa, or enlargement of the spleen, together with the irregularity of the temperature is usually sufficient to distinguish it.

The prognosis is unfavourable; and little can be done in the way of treatment beyond that indicated in any acute fever.

CANCER—CARCINOMA.

In cancer we find a general malnutrition associated with local new growths of a distinctive kind. It yet remains to be settled whether, on the one hand, the new growth is but the local expression of a constitutional taint; or, on the other, whether it is primarily a local disorder and only affects the general system secondarily. A cancer is said to be *malignant* because it tends to grow indefinitely, to implicate the neighbouring lymphatic glands, to appear in distant parts as a "secondary deposit," and to recur after removal.

The essential feature of a cancerous growth consists of epithelial cells imbedded in the meshes of a fibrous stroma. The character of the *cells* varies. They broadly resemble the epithelium peculiar to the epithelial surface from which the cancer originates, and hence may be flat, spheroidal, or columnar. The *stroma* varies greatly in amount, and consists of bands of fibrous tissue, interspersed with connective-tissue cells, arranged so as to enclose alveolar spaces of variable size, in which the epithelial cells are contained. The *blood-vessels* are confined to the stroma, and in no case encroach upon the alveoli. They are accompanied by *lymphatics* which communicate directly with the alveoli.

As already said cancer originates from some epithelial surface such as that of the skin or mucous membrane, or in the acini of some gland. The cells at first proliferate, and then extend by club-shaped processes into the subjacent connective-tissue, irritating it into more or less active growth (stroma). The margins of the growth are diffuse and ill-defined in primary cancer as a rule; but secondary cancer has more often a well-marked boundary. When it grows slowly, and the meshes of the stroma are small, it is hard (*scirrhus*); when it grows rapidly and the meshes are large, it is softer (*encephaloid*); when it grows from the skin it is called an *epithelioma*, the horny cells of which are arranged in concentric layers, forming *nests* on cross-section. The commonest seats of primary cancer are the skin, mamma, pylorus of stomach, rectum, uterus, kidney, and gullet.

Secondary cancer is due to epithelial particles of the primary growth becoming detached and carried away by the blood-vessels or lymphatics. Embolic impaction follows, and a new and independent growth is started, similar to that from which the embolus came. Small cells are more easily carried than large ones; therefore—*cæteris paribus*—those cancers which have the largest cells (*e.g.*, cancer of skin) are the least malignant; and *vice versa* (*e.g.*, cancer of the mamma).

Cancerous growths are very liable to undergo fatty and colloid degeneration. The latter is especially common in cancer of the alimentary canal. Ulceration is also of frequent occurrence, leading to unhealthy intractable sores.

Cancer is undoubtedly hereditary, though it is sometimes met with in the absence of any family history of the kind. It rarely occurs until after middle age, but the young are not altogether exempt. Females are more prone to it than males, but this would appear to be largely due to the frequency with which the mamma and uterus are affected.

SYMPTOMS.—The constitutional effects of cancer are manifested by what is termed the *cancerous cachexia*, characterised by a peculiar sallowness of complexion, an anxious expression, pinched features, and general emaciation, together with more or less prostration, debility, and languor.

Pain (usually of a burning or lancinating kind) and tenderness of the affected part, together with the physical signs of a "tumour," are almost always present. Other local symptoms are apt to arise from direct or indirect interference on the part of the growth with the surrounding structures.

The diagnosis, prognosis, and treatment, will be referred to when describing the disease as it affects special parts.

ANÆMIA.

THE term anæmia is one which is very loosely employed, but speaking generally, it may be applied to any functional disturbance or defective nutrition which is attended with a depreciation in the physiological value of the blood. The number of red blood cells is often (though not necessarily) reduced, and it is common to find many which are small and deformed. There is also a deficiency of hæmoglobin and albuminates of the blood, together with an increase in the percentage of saline, and (usually) also of watery constituents.

The terms *spanæmia* (thin blood), *hydræmia* (watery blood), and *oligæmia* (scanty blood) are sometimes employed in place of *anæmia*.

It would be impossible to enumerate all the various causes of anæmia. They may, however, be classified in three groups:—(1) *Deficient supply of the proper materials for the formation of healthy blood*, such as good food, fresh air, light, &c.; (2) *excessive drain upon the nutritive constituents of the blood* by hæmorrhage, over-exertion, long-continued discharges, &c.; and (3) *general malnutrition* arising, *e.g.*, from dyspepsia, organic disease of various kinds, fever, chronic metallic poisoning, &c. Anæmia is common to both sexes, and to all periods of life.

SYMPTOMS.—In chronic anæmia, the integuments are of a pale, sallow, or waxy hue, which is especially well marked on the mucous membrane of the mouth and eyes. Œdema of the ankles, legs, or eyelids, is of frequent occurrence, and sometimes affects a much wider area.

The general nutrition suffers in proportion to the duration and intensity of the anæmia. The skin becomes thin, and (when not œdematous) wrinkled; the subcutaneous fat disappears; the muscles waste and get flabby; the extremities are chilly; the scalp becomes scurfy, and the hair falls off, producing more or less temporary baldness; the nails also are thin, and often ill-formed.

Nervous symptoms are uniformly present, and are in the main characterised by exalted irritability, followed by speedy exhaustion. Thus, the anæmic patient is fretful, querulous, whimsical, and lacks decision; his memory is bad, he is disinclined for exertion, soon gets tired, and yet sleeps badly. Neuralgic pains (especially in the left side) and twitching or trembling of the muscles are often met with.

Palpitation and shortness of breath occur on the least exertion. A systolic murmur of maximum intensity in the left second intercostal space over the pulmonary artery is often heard; and a continuous venous hum (*bruit de diable*) over the large veins at the root of the neck is equally common. In some cases, systolic murmurs may also be heard at the apex of the heart, or at the aortic orifice. These so-called *hæmic* murmurs result partly from an altered condition of blood, and partly from the physical conditions of the parts in which they are produced. The pulse is usually frequent and soft.

The appetite is poor or lost; digestion is always feeble, and is accompanied with pain and flatulence. The bowels are usually constipated.

The urine is copious, pale and limpid. Menstruation is generally disturbed, being scanty, absent, or painful. Leucorrhœa is of frequent occurrence.

TREATMENT.—The first consideration is to ascertain the cause and to remove it if possible. General treatment is of great importance. Thus, fresh air, out-door exercise, plenty of sleep, and tepid or cold bathing, greatly aid recovery. Since digestion is very feeble, the food should be light and nutritious, taken in very moderate quantity, and at shorter intervals than usual. A little light wine or sound ale is often of service, but should never be taken without food.

Iron in some form or another is the most important medicinal remedy. The blander preparations (*e.g.*, ferri et ammon. cit.) are usually preferable at first, and may often be advantageously combined with strychnia. The tendency to constipation often calls for the addition of some aperient to the iron, of which aloes is the best. A convenient formula is the pil. aloes cum ferro (B.P.); or a combination of sulphate of iron with sulphate of magnesia. Iron in any form is apt to disagree where the tongue is foul, and the stomach much disturbed. Under such circumstances it is better to begin with a purgative, followed by a short course of an alkali (or mineral acid as may be indicated) combined with some vegetable bitter. Among other remedies may be mentioned arsenic (liq. arsenicalis) and phosphate of lime. The former I have often found of great service in anæmia recurring as the result of hæmorrhage; and the latter is recommended by Dr. Ringer in the anæmia of rapidly growing children, or women weakened by over-suckling, &c.

In anæmia dependent on syphilis, or metallic poisoning, and the like, the treatment must be directed to the condition on which the anæmia depends.

CHLOROSIS.

Chlorosis, though in some respects resembling anæmia, differs from it in many particulars. The characteristic feature of the blood-change consists in a marked deficiency of hæmoglobin, without any diminution in the number of red corpuscles. The quality of the plasma remains unchanged. It comes on in young girls about the age of puberty, and does not admit of being traced to familiar causes, like anæmia. The pathology of chlorosis is very obscure.

The *symptoms* are in the main similar to those of anæmia, but are indicative rather of functional disturbance, than of malnutrition. The pallor is intense, and of such a peculiar greenish wax-like kind, that the disease is popularly known as the *green-sickness*. The body does not waste; on the contrary, there is sometimes a tendency to obesity, which ceases on recovery from the disorder. Œdema is rare, and when present is inconsiderable. Menstruation (if it has appeared) ceases, or its first appearance is delayed till after recovery. In exceptional cases, however, the menstrual flow is not checked. It is remarkably amenable to treatment by iron (far excelling anæmia in this repect), but the drug must be given in *large doses*. The dose is of more importance than the form of preparation; but the most convenient and satisfactory, in my experience, is a pill of five grains of *dried* sulphate of iron, made up with glycerine of tragacanth. Three of these pills (increased up to six or eight) may be given daily.

PERNICIOUS ANÆMIA.

The term "pernicious anæmia" is applied to a very fatal form of idiopathic anæmia, the origin of which is quite obscure. It differs from chlorosis in that it affects males as often as females, that it occurs later in life—generally between 25 and 40 years of age, that it is not curable by iron, and that it is nearly always fatal. The blood is very pale and watery. The red blood-cells are always reduced in number, sometimes even to a tenth of the normal amount, and many small spherical dark cells are often seen. The white cells may be in slight numerical excess. The total amount of hæmoglobin is much reduced, though the amount *per* corpuscle is increased. There is also marked fatty degeneration of the heart, and of the small arteries and capillaries. The spleen may be slightly enlarged, and the bone-marrow is unusually dark-coloured. Hæmorrhages are common, especially in the retina and serous membranes.

Symptoms.—The patient is pale and presents all the usual features of extreme anæmia. The temperature is variable, but is generally raised by two or three degrees. Retinal hæmorrhages are of common occurrence, and may be seen with an ophthalmoscope. The urine is dark-coloured, and is sometimes albuminous. The progress of the disease is steadily unfavourable, and death follows in a large proportion of cases. Its usual duration is from six to twelve months.

The *treatment* is almost hopeless. Iron is of no use, but arsenic—steadily pushed—has sometimes proved beneficial. Transfusion has been followed by recovery in a few cases.

HÆMOPHILIA.

This morbid condition is characterised by a tendency to profuse bleeding on the slightest provocation, such as the extraction of a tooth, or an accidental wound of any kind. Bleeding from the mucous surfaces, especially from the nose, is common from causes so slight as often quite to escape notice. Again, subcutaneous hæmorrhages are apt to occur after trifling injury, causing bruises or deep-seated extravasation.

All instances of the disease are not equally severe. Legg recognises three groups:—(1) where there is a tendency only to bruising readily or spontaneous ecchymoses; (2) cases of bleeding from mucous surfaces only; and (3) liability to severe bleeding of every kind.

Apart from anæmia directly due to loss of blood, and the troubles attributable to it, the general health may be good between the attacks of bleeding. Occasionally, however, rheumatic inflammation of the larger joints is observed, especially of the knee. More than half of those afflicted with hæmophilia die before puberty.

This curious affection is strikingly hereditary, and is much more common in males than in females. The latter may and often do transmit it, without themselves suffering from bleeding. Little or nothing is known as to its pathology; nor can it be arrested by treatment.

LEUCOCYTHÆMIA.

Leucocythæmia is the term applied to a morbid and persistent increase in the white corpuscles of the blood, which is associated with enlargement of the spleen and sometimes of the lymphatic glands.

The causation of the disease is very obscure, it affects men much more than women; and though it may occur at any age, it is much more frequent during middle life.

Morbid Anatomy.—The colour of the blood is obviously paler than normal, and the proportion of white to red blood-cells is enormously increased, the former being sometimes even more numerous than the latter.

The spleen is always enlarged from overgrowth of its pulp; and in about a third of all cases the lymphatic glands *subsequently* become enlarged; while small local overgrowths of lymphoid tissue may also be found in the substance of the liver, kidneys, lungs, and elsewhere.

Typical cases of Hodgkin's disease (lymphadenoma) differ from leucocythæmia in that the white blood-cells are not in excess, enlargement of the lymphatic glands always exists from the first, enlargement of the spleen is not constantly present, and when it occurs is not so marked. A certain proportion of cases of Hodgkin's disease ultimately become leucocythæmic.

Clinical History.—Apart from the enlargement of the spleen and lymphatic glands, we meet with slowly progressive anæmia, weakness, and emaciation, with incapacity for exertion, shortness of breath, and digestive derangements. The temperature is often somewhat raised, and sometimes persistently so. The urine is normal in appearance; but the urea is diminished, while the uric acid and urates are increased. In the later stages, there is a tendency to dropsy, and hæmorrhage from various surfaces. The prognosis is exceedingly grave, and death occurs sooner or later, more often from asthenia, but sometimes from severe hæmorrhage, or some complication.

The *treatment* of leucocythæmia is in the highest degree unsatisfactory, and is limited purely to palliative measures.

DIABETES MELLITUS.

Diabetes is characterised by the persistent presence in the urine of grape-sugar (glucose) in more or less abundance; and is attended with constitutional disturbance of a marked kind. Normal urine contains, it is said, a very minute trace of sugar, and we may meet with temporary glycosuria in various morbid conditions, but such are not included in the affection now about to be described.

Diabetes is occasionally hereditary; and while it occurs at all ages, it is much more frequent in the male than in the female. Exposure to cold, violent emotion, injury or disease of the nervous system are among the chief exciting causes, but very often none can be traced.

The *pathology* of diabetes is very obscure, and it is probable that it is not identical in every case. We may, however, pretty safely conclude that it is closely related to some disturbance of the glycogenic functions of the liver. In some cases the glycogenic activity of the organ would appear to be increased, and more sugar is thus thrown into the circulation than can be metamorphosed in the tissues, the surplus being excreted with the urine. Whether this increased activity is due to direct nervous influence upon the hepatic cells, whether it arises from vaso-motor disturbance producing dilatation of the hepatic arterial system, or whether it is sometimes the one, and sometimes the other, future observation must be left to decide. Other cases, again, in which the urine becomes normal by avoiding all saccharine or starchy food-stuffs, may be explained by assuming inadequate conversion into glycogen of the saccharine matter which is absorbed from the alimentary canal, an excess of sugar thus appearing in the blood. No characteristic morbid appearances are met with.

Symptoms.—The onset is generally insidious, and attention is first aroused by gradually increasing weakness in spite of a wonderfully good appetite. Simultaneously the patient may note an increase in the quantity of urine passed, but this is frequently not observed till attention has been attracted to it by the doctor. But however the condition first attracts attention, the essential features, when fully established, are—excessive abundance of saccharine urine, intense thirst, voracious appetite, progressive emaciation, dryness of the skin, with various functional disturbances, ultimately followed by death.

The urine.—The quantity is largely increased, and varies from 5 to 12 pints daily, or even more. It is acid, clear, and of remarkably brilliant aspect. Its sp. gr. is unusually high, and varies from about 1030 to 1060, a fair average being about 1040. It contains a large but variable quantity of sugar, usually ranging from 15 to 25 oz. daily (8 to 12 per cent.); but may be much more or much less.* It is most abundant after meals, especially after saccharine or starchy food; and sometimes disappears altogether when these are withdrawn. The amount of urea is increased; and a little albumen is sometimes present. Micturition is more frequent.

Thirst is always excessive, the quantity of fluid drunk roughly corresponding with that of the urine passed. The *appetite*, too, is generally—but not always—voracious.

Emaciation is progressive from the first; and in the later stages is extreme, and affects all the soft tissues. It must not be forgotten that some diabetic patients are excessively corpulent, especially those of a gouty habit.

The *skin* is dry, harsh, irritable, and often œdematous about the ankles. Certain eruptions—especially boils—are common. The *tongue* is red and dry. The teeth are apt to decay rapidly, while the gums recede, and become unhealthy.

Functional Derangements.—Palpitation with shortness of breath on exertion, digestive disturbance, flatulence, and constipation of the bowels; mental dulness and want of resolution; weak or absent sexual desires; visual troubles with or without organic change (cataract is not infrequent), are among the more important functional troubles.

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* *For the tests for sugar in the urine, refer to the chapter on "Urine."*

As to *complications*, the chief is pulmonary phthisis, which supervenes in at least half of all cases, and is a frequent cause of death. A peculiar form of collapse, known as diabetic coma, is also not uncommon, and is very fatal. The patient (often after some unusual fatigue) becomes weak and apathetic, the respiration is slow and sighing; the breath has a curious ethereal smell; coma gradually develops; and death occurs in from one to three days. Low forms of pleurisy, pneumonia, peritonitis, abscesses, carbuncle, and gangrene are also occasionally met with.

The course of diabetes is slow, chronic, and progressive, the duration varying from one to three years. Sometimes, however, it runs an acute and rapid course. Recovery is exceptional, and death usually occurs either from asthenia, coma, or some complication.

Glycosuria is not uncommon in gouty subjects, and sometimes alternates with a sandy deposit of uric acid, the sugar temporarily disappearing. In such cases the usual symptoms of diabetes are either absent or ill-marked, and the course is very chronic.

TREATMENT.—This may be discussed under three heads: general, dietetic, medicinal.

General.—Exercise (always stopping short of fatigue), fresh air, warm-clothing, and occasional baths are of considerable benefit. The bowels should be carefully regulated by castor oil, or saline aperients. The digestive functions require attention and for this purpose nothing is better than alkalies combined with some vegetable bitter. To relieve the intense craving for food, Sir Wm. Roberts recommends a pill containing two or three grains of asafœtida twice or thrice daily. Sleeplessness may be combated with opium, bromide of potassium, and the like.

Dietetic.—All saccharine and starchy foods should be avoided; *e.g.*, sugar, bread, rice, tapioca, potatoes, peas, beans, carrots, turnips, parsnips, alcohol, and most fruits. The diet should be selected from the following: milk, butter, cheese, eggs, meat, fish, and green vegetables. Any bread used should be well and uniformly toasted; or gluten bread, bran-cakes, or almond biscuits may be substituted for it. Donkin speaks highly of a dietary consisting exclusively of six to eight pints of skimmed milk daily. Acidulated drinks, or a solution of bitartrate of potash, are of service in relieving the extreme thirst.

Medicinally, opium appears to be the most valuable drug, and should be given in small doses (gr. $\frac{1}{2}$ *ter die*) at first, gradually increased. Others prefer codeia to opium, which is given in similar doses. Opium, however, is of little use in gouty forms of diabetes, and is sometimes positively harmful. In such cases great benefit may be occasionally derived from the administration of salicylate of soda (℈j. *ter die*). The anæmia and emaciation often call for iron and cod-liver oil. Gouty diabetes is sometimes much benefited by alkalies, or a course of treatment at Carlsbad.

ADDISON'S DISEASE.

It is difficult to know under what class this affection ought to be described. For the present purpose it may be regarded as a constitutional malady, characterised by general debility, bronzing of the skin, and cheesy changes in the supra-renal capsules. The large sympathetic ganglia in the upper part of the abdomen are also said to exhibit changes of a degenerative nature.

The prominent *symptoms* are (1) general and progressive debility; (2) pigmentation of the skin; and (3) pain in the epigastrium or loins, with nausea or vomiting.

The *onset* is insidious, and is marked by increasing weakness (with little or no emaciation), shortness of breath, palpitation, a feeble frequent pulse, lassitude, languor, and prostration. The *skin* meantime becomes darker, with a tint of yellowish brown, or greenish black of variable depth. This change usually begins on the surface of the upper half of the body; and is most intense in those parts which are naturally darker than elsewhere, *e.g.*, the lower eyelids, axillæ, areolæ of nipples, and pudenda. The conjunctiva remains white and pearly. Black spots sometimes form on the lips or on the buccal mucous membrane. In addition there is as a rule more or less pain in the epigastrium or loins, with nausea or even severe vomiting.

Remissions not infrequently occur, but are only of temporary duration, the progress being invariably towards a fatal termination, though slow and tedious. Death ultimately occurs from asthenia or syncope, and is sometimes preceded by grave nervous disturbance.

As to *treatment*, all that is possible is to endeavour to keep up the strength by tonics, cod-liver oil, wine, good food, warmth and fresh air.

SECTION IV.

DISEASES OF THE RESPIRATORY SYSTEM.

TABULAR ARRANGEMENT OF PHYSICAL SIGNS.

I. INSPECTION.

- 1. ALTERATIONS IN THE SHAPE OF CHEST . .
  - Congenital, or due to pre-existing disease . . . .
    - Alar chest.
    - Flat chest.
    - Pigeon-breast.
    - Transverse constriction.
    - Rickety chest.
  - Due to existing disease . . . . .
    - Enlargement or bulging
      - Bilateral.
      - Unilateral.
      - Local.
    - Diminution or Shrinking
      - Bilateral.
      - Unilateral.
      - Local.
- 2. ALTERATIONS OF RESPIRATORY MOVEMENTS . .
  - Increased.
  - Diminished.
  - Irregular.
  - Laboured.
  - Thoracic movements in excess.
  - Abdominal movements in excess.

II. PERCUSSION.

- PERCUSSION-NOTE.
  - Resonance . . . .
    - Hyper-resonance.
    - Tubular resonance.
    - Amphoric resonance.
  - Dulness . . . . .
    - From slight deficiency to absolute dulness.
  - Peculiar sounds . .
    - Metallic resonance.
    - Cracked-pot sounds.

III. Palpation.

- Fremitus . . . (Vibration of chest-walls as felt by hand).
  - Vocal fremitus . .
    - Increased.
    - Diminished.
    - Absent.
  - Friction fremitus.

IV. Auscultation.

- 1. Breath-sounds altered as to . . . .
  - Intensity . . . .
    - Increased, exaggerated, or puerile.
    - Diminished, or weak.
  - Rhythm . . . .
    - Jerky, irregular, or wavy.
    - Expiration prolonged.
  - Quality . . . . .
    - Harsh.
    - Bronchial.
    - Tubular.
    - Blowing.
    - Cavernous.
    - Amphoric.
- 2. Rales or Rhonchi . . .
  - Sibilant.
  - Sonorous.
  - Mucous.
  - Crepitation . . .
    - True.
    - Redux.
  - Crackling . . . .
    - Dry.
    - Moist.
  - Hollow . . . . .
    - Amphoric.
    - Metallic.
    - Cavernous.
- 3. Vocal Resonance.
  - Increased or modified . . . . .
    - Bronchophony.
    - Pectoriloquy.
    - Œgophony.
  - Diminished.
- 4. Pleural Friction.

GENERAL SYMPTOMATOLOGY.

Alterations in the Voice.—True vocal sounds originate in the larynx, and are due to the concurrence of an expiratory effort with approximation, tension, and vibration of the vocal cords. From feebleness of the expiratory act, the voice becomes faint and weak; and from uncertainty in its production, the voice becomes tremulous. Should paralysis prevent the due approximation or tension of the cords *aphonia* (loss of voice) results. If the vibration of the cords is interfered with by inflammation, ulcers, or new growths, hoarseness or even aphonia may occur. An alteration in the voice, short of absolute tonelessness, is often called *dysphonia*.

Temporary variations in the vocal *pitch* depend on alteration in the tension of the cords, and their relaxation by inflammation or otherwise therefore deepens its tone. The *quality* of the voice is mainly related to the physical condition of the throat and mouth, it becomes modified by enlarged tonsils, paralysis of the soft palate, interference with the movements of the tongue, &c.

Alterations of Respiration.—*Dyspnœa* is a term which is applied both to rapid and laborious breathing. The depth of respiration is generally (but not always) in inverse proportion to its frequency. Dyspnœa is often due merely to excessive nervous irritability, such as that met with in hysteria. Far more often, however, it is the proximate result of an abnormal condition of the blood which is supplied to the respiratory nervous centre in the medulla. Thus, dyspnœa occurs (1) when the blood is overheated, as in fevers; (2) when it contains unusual ingredients, as in some cases of uræmia; (3) when it is deficient in oxygen, as a result of anæmia, disease of the lungs or heart, obstruction of the air-passages, mechanical interference with the movement of the chest-walls or diaphragm, paresis or spasm of the respiratory muscles.

Sometimes the difficulty is mainly attached to inspiration as in certain forms of laryngeal paralysis; at other times to expiration, as in asthma; but more often it affects both inspiration and expiration. When dyspnœa is so severe that breathing is only possible in a more or less upright posture, it is termed *orthopnœa.* When the difficulty in breathing comes on in more or less sudden attacks of temporary duration, it is said to be *paroxysmal.* In all cases of partial obstruction or constriction of the laryngeal or tracheal passages, the breath-sounds have a peculiar harsh wheezing quality, known as *stridor* or *stridulous breathing.* Another variety, known as *Cheyne-Stokes' respiration,* is characterised by a series of respirations gradually increasing in depth up to a maximum, then gradually declining to a minimum, and followed by a considerable pause. It is a sign of very unfavourable import.

The respiration is slowed in syncope, collapse, and in various lesions of the nerve centres.

Cough.—Coughing consists of a long drawn inspiration, followed first by closure of the glottis, and then by one or more forcible expiratory efforts by which the glottis is forced open with noise. It is a purely reflex act, the afferent impulses of which travel upwards for the most part through fibres of the vagus nerve coming from the throat, air-passages, lungs, stomach, or ear. Irritation of any of these parts is therefore liable to be attended with cough. Cough may be simple or paroxysmal; as to quality, it may be hacking, hoarse, barking, metallic, aphonic, and so on; and it may be dry, or accompanied with expectoration.

Expectoration.—In health, expectoration is rarely necessary, but when the secretion of mucus in the air-passages is free, it is expelled as a *mucoid* sputum, and may be more or less frothy from mixture with air. Pus or blood from the air-passages, or from parts in communication with them, may be expectorated either in a pure state or mixed with mucus. Viscid purulent-looking lumps, suspended in mucoid sputa (*nummulated sputa*), are highly suggestive of dilated bronchi or phthisical cavities; and such sputa are often very offensive. This odour must be distinguished from that of gangrene. Fibrinous moulds of the smaller air-passages are sometimes expectorated.

Sub-Section I.

GENERAL RESPIRATORY DISEASES.

WHOOPING-COUGH.

Whooping-cough or Pertussis is a specific infectious and often epidemic disease. The poison on which it depends has not as yet been definitely ascertained; but whatever its nature it is contained in the sputa, and may be carried to others by *fomites*, such as clothing, articles of furniture, and the like. Speaking generally, its effects are manifested by catarrh and hyperæsthesia of the air-passages. It is essentially a disease of childhood, but adults are not entirely exempt from it. A second attack is rare.

The anatomical appearances are those of bronchial catarrh, with enlargement of the bronchial glands.

Symptoms. — Three stages are usually recognised. 1. The *catarrhal stage* begins gradually with fever and the symptoms of an ordinary catarrh, and is attended with cough and abundant viscid expectoration. It continues for a very variable period, but its average duration is about ten or twelve days. 2. As the *convulsive stage* sets in, the catarrhal symptoms often abate, but the cough gets worse, lasts longer, and assumes a characteristic paroxysmal form. Each attack begins with a long-drawn inspiration, followed by a *series of sharp, short, rapidly successive expiratory coughs*, and concludes with a prolonged crowing inspiration or "whoop." The paroxysm may be repeated once or twice until at last some glairy mucus is expelled, or actual vomiting occurs, and the breathing once more becomes quiet. During the attack the face is dusky, the veins become swollen, the eyes suffused, suffocation seems imminent, and the breath-sounds on auscultation are feeble and suppressed. Hæmorrhages from the nose, mouth, ears, or beneath the conjunctivæ are apt to occur. The number of paroxysms *per diem* varies widely, but they are usually both worse and more frequent at night. 3. After the lapse of three or four weeks the *stage of decline* gradually sets in, the coughing-fits become fewer and less severe, the expectoration more easy, the sputa muco-purulent, until convalescence is re-established. The physical signs—in the absence of pulmonary complications—are unimportant.

Simple pertussis, though often protracted, is rarely fatal; but it is quite otherwise when complicated. Whooping-cough may supervene upon other diseases. especially *measles;* or it may be complicated (1) with *bronchitis*, *pulmonary collapse*, and *pneumonia ;* (2) with *convulsions* and *grave nervous symptoms ;* or (3) with *disordered conditions of the stomach and bowels.*

Diagnosis.—The peculiar cough and expectoration are quite characteristic.

Treatment.—During the *first stage*, confinement within doors, some simple medicine such as vin. antim. or vin. ipecac. with paregoric, warm baths, and counter-irritation to the chest are necessary. In second or *spasmodic stage*, the more important of the many drugs recommended are bromide of potash, belladonna (well borne by children), hydrocyanic acid (♏½-j.), lobelia (♏v. of tinct. every hour), alum (gr. ij.-vj. every three hours), chloral hydrate, morphia, chloroform, and carbolic acid. Counter-irritation with sinapisms or stimulating liniments is of service. During the *decline* cod-liver oil, tonics, and change of air are urgently required. Each case, however, must be treated according to its special type, and complications dealt with as they arise.

The diet should be light and unirritating; and the secretions require attention.

ASTHMA.

Asthma may be briefly defined as *paroxysmal dyspnœa*. The *paroxysm* may be preceded for a variable time by a sense of oppression and constriction of the chest, with wheezy respiration, or in some cases by the abundant discharge of pale watery urine. On the other hand it often comes on without any warning, and most commonly at night. The patient awakes scarcely able to breathe, adopts the sitting or erect posture with the shoulders raised and fixed, the head thrown back, and the mouth open, and all the extraordinary muscles of respiration are brought into play. The face wears an aspect of terror; the eyes are widely opened; the skin is pale and dusky, and often bedewed with sweat; the extremities are cold; and the pulse is small and quick. The breathing, however, is not hurried, but inspiration is short and jerky, and expiration is inordinately prolonged.

The resonance of the chest on percussion is increased, vesicular breath-sounds are weak or even suppressed. and attended with dry sibilant râles. These physical signs very commonly exhibit a patchy distribution, which is frequently shifting.

Towards the end of an attack, cough occurs with expectoration of small, firm, non-aerated pellets of mucus, in rare cases mixed with blood. The *duration* varies greatly; the paroxysm may either pass off in a few minutes, or continue on and off for days. When it lasts long, or is left to itself, it subsides gradually; but if short, or arrested by treatment, it often ends abruptly. The expectoration often continues for several days afterwards. Asthma sometimes assumes a *periodic* character, which is liable to change its type as the case proceeds. It rarely begins before 20 or after 50 years of age. Persistent severe asthma is very apt to lead to emphysema. Bronchitis also is a common complication.

Etiology and Pathology.—It has been usually taught that while some cases of asthma are *symptomatic* or secondary to other morbid conditions, such as pulmonary, cardiac, or renal disease, there are cases in which it occurs independently of such causes, and are therefore termed *idiopathic* or *nervous*. Such a distinction seems to introduce an unnecessary confusion into the subject. The most important facts, in the writer's opinion, appear to be these. In the first place the suddenness both in the appearance and disappearance of the attacks unquestionably points to a nervous origin. According to this view, asthma is a neurosis, affecting the nervous centres in the medulla which preside over the respiratory mechanism, and the blood-supply of the lungs. A special excitability of these centres, and a liability to disturbance, either inherited or acquired, must be assumed as a primary factor in the etiology of asthma under all circumstances. Authorities mainly agree in attributing the immediate cause of the attack to obstruction in the finer bronchi. Two conditions are put forward as responsible for such obstruction—spasm of the bronchial muscular coat, and engorgement with swelling of the respiratory mucous tract through vaso-motor agency. As to their respective importance, or their relationship to each other, there is wide difference of opinion, and the matter is as yet unsettled. It still remains to consider the circumstances which lead up to and predispose to an asthmatic paroxysm. Perhaps the simplest cases are those which depend upon the introduction of dust, pollen, or some other mechanical irritant into the air-passages. In others, the exciting cause may be structural change in the lung-tissue. In a third group—the supply of mal-aerated blood to the medulla. In a fourth group—a deficiency in the blood-supply of the medulla by vaso-motor spasm. In a fifth group—peripheral irritation from disease of the stomach, bowels, uterine organs, or from worms. In a sixth group, there may be direct irritation of the medulla by impure blood as in uræmic asthma, or by the action of certain drugs as ipecacuanha. The special liability to attacks during sleep may be probably explained by depressed respiratory activity at this time, and by confinement—as too often happens—in close and impure air.

The *diagnosis* rests upon the paroxysmal and usually sudden nature of the attacks, with more or less complete recovery in the intervals between them.

Treatment.—*During the paroxysm.* Remove tight clothing and give fresh air. Medicinally, hypodermic injection of morphia, the administration of chloral or lobelia (tinct. ♏xxx. upwards every half hour), the inhalation of the smoke of tobacco stramonium or nitre paper, emetics—of which ipecacuanha is the best, inhalation of chloroform, and strong black coffee have been chiefly recommended.

During the interval. Special attention should be given to the conditions on which the attacks appear to depend. Change of air is often necessary. Tonics, as quinine, steel or arsenic, combined or not with antispasmodics, as lobelia, are of great service.

SUB-SECTION II.

DISEASES OF THE AIR-PASSAGES.

NASAL CATARRH.

ACUTE NASAL CATARRH or coryza is most frequently part and parcel of a simple catarrh from chill ("*cold in the head*"). But it also occurs from local irritation of any kind, or in connection with certain acute diseases, or from the presence of a polypus. When due to local causes the symptoms are mainly confined to the nose, and consist of obstruction of the nasal passages from swelling of the mucous membranes, nasal voice, local discomfort or pain, and sneezing and frontal headache from reflex irritation. In the early stages, there is more or less clear watery discharge, which afterwards becomes mucoid, and during convalescence is mucopurulent. When brought about by cold, there is in addition general malaise, slight fever, and the catarrhal process is apt to also involve the throat, trachea, and alimentary canal. In frequently recurring catarrh, the possibility of some nasal growth should always be kept in view.

A hot bath followed by a moderate opiate at bedtime, and a saline purge next morning will often abort a simple "cold," if resorted to early. A few drops of spirit of camphor on sugar may be substituted for an opiate if the latter be thought undesirable. If there be much fever, tinct. aconiti in drop doses every hour is of great service. When the catarrh is established, saline diaphoretics are indicated; and during convalescence tonics are required.

Chronic nasal catarrh is generally met with as a sequel of previous acute attacks, and its etiology is practically the same. In weak states of health, particularly in connection with a strumous or syphilitic taint, catarrhal inflammation is apt to be very difficult to "shake off;" and, in cases of long-standing, hypertrophy, atrophy or ulceration of the mucous membrane, or necrosis of the nasal bones may occur.

The local symptoms are essentially similar to those of acute catarrh. The character of the discharge, however, varies. Sometimes it is abundant and watery, especially where there is much nervous irritability of the cavities. In other cases it is more viscid, and either mucoid or muco-purulent. Under certain circumstances (not properly understood), the discharge is quite scanty, and dries on the inner surface of the nose cavity; forming flakes which undergo decomposition and emit a disgusting and characteristic smell, hence the term *ozæna* (Ὄζη, a stink) is applied to it. It is a fortunate thing that the patient usually loses his sense of smell. It should be remembered that ozæna occurs quite independently of ulceration or necrosis, though of course these conditions are accompanied, as elsewhere, with a bad odour.

As to treatment, unless the cavities are extremely irritable, washes or injections are necessary. Dilute tepid solutions of bicarbonate of soda, borax, or common salt, (about gr. x. or xx. to the ounce) are of most service. If special detergent effects are required, the addition of a little carbolic acid is desirable. Mild astringent solutions are also valuable in certain cases, *e.g.*, zinc sulphate (gr. ss.), alum (gr. iv.), or tannin (gr. iv. to the ounce). Sprays may be substituted for injections if the latter are not tolerated, but they are less effective. The insufflation of certain powders *(see Therapeutic index)* has also been recommended. Frontal pain may be relieved by local use of morphia ointment (gr. ij. to the ounce).

Constitutional treatment of a tonic kind is very necessary, with change to a warm and dry climate if possible. Again, a caution is necessary to avoid overlooking the presence of nasal growths.

ORGANIC DISEASES OF THE LARYNX.

Organic diseases of the larynx include acute and chronic inflammations together with the conditions to which inflammation gives rise, such as ulceration, œdema, and perichondritis; and new growths.

Certain local phenomena are more or less common to all laryngeal affections. (1) *Alterations in the voice.* These vary from slight huskiness to complete aphonia; together with modifications of its quality and pitch. (2) *Dyspnœa.* This is noisy, hissing, or stridulous in proportion to the extent of obstruction in the air passages by which it is caused. Sudden attacks of a paroxysmal nature are frequently met with, as a result of spasmodic closure of the glottis. Inspiration and expiration are equally affected when the obstruction is material and persistent; but in certain paralytic and other conditions, either inspiration or expiration may be alone affected. (3) *Cough* due to laryngeal irritation is frequent, paroxysmal, and of a harsh, hoarse or barking quality. (4) The *expectoration* in recent inflammation is clear and scanty, and viscid; but later on and in chronic laryngeal affections it is more abundant, muco-purulent, or it may be streaked with blood. (5) *Dysphagia*, or difficulty in swallowing is not a common symptom, but is apt to occur when the epiglottis is the seat of disease, especially in connection with ulceration. (6) *Pain* is frequently present and varies from mere uneasiness, and a sensation of tickling about the throat, to burning irritation; and it is sometimes accompanied with external tenderness on pressure of the adjacent parts.

Acute Catarrhal Laryngitis.

Among the most frequent causes of acute catarrhal laryngitis are (*a*) catching cold, (*b*) the introduction of irritating vapours acid liquids or water into the larynx, (*c*) over-straining the voice, and (*d*) morbid growths and ulceration. It moreover occurs as a frequent symptom in many acute diseases, such as scarlet fever, measles, typhus, and small-pox. Previous attacks, enlarged tonsils, post-nasal growths, and sedentary indoor occupations may be regarded as predisposing causes. It is more common and more serious in children than adults.

Symptoms.—As a rule (but not invariably) the disorder begins with the signs of an ordinary cold. There is more or less febrile disturbance accompanied with hoarseness of the voice and cough, sometimes increasing to aphonia. Burning or tickling sensations in the larynx may be complained of, and the throat may be sore. The character of the expectoration—if there be any—varies greatly. With the laryngoscope, the mucous membrane is seen to be red and turgid. In children, from the small size of the laryngeal passage and the greater laxity of the mucous membrane, there is relatively more swelling, and consequently more interference with breathing, than in adults. Children are also prone to acute paroxysms of dyspnœa and stridor from spasmodic closure of the glottis. In this respect there is considerable resemblance to croup, with which disorder catarrhal spasmodic laryngitis is often confounded. The attacks take place most frequently at night; but towards morning there is usually a well marked remission, and during the day the child is much better. For one or two nights the paroxysms may recur, but with diminished severity; and under appropriate treatment the affection generally subsides in the course of a few days. Some hoarseness often remains behind for a long time; and, after one attack, recurrence is apt to take place on every slight chill.

Acute œdema of the larynx is also an occasional and dangerous complication of catarrhal laryngitis. It happens most often in connection with scarlet fever, erysipelas, diffuse inflammation of the neck, and the introduction of scalding water into the larynx. The œdema mainly involves the loose tissue of the ary-epiglottic folds, which may become so swollen as entirely to block up the entrance into the larynx. Under these circumstances, severe dyspnœa is inevitable, and if not relieved, death from asphyxia soon follows. Thus although acute catarrhal laryngitis usually ends favourably, yet dangerous complications may occur which demand prompt attention. Sometimes it passes into the chronic variety.

ACUTE MEMBRANOUS LARYNGITIS—CROUP.

The relationship of croup to diphtheria has long been a matter of controversy. For the present, croup must be regarded as a clinical term, indicating a peculiarly fatal disease of childhood, characterised by membranous laryngitis. The false membrane which is formed on the mucous membrane of the larynx and adjacent parts is in most cases due to diphtheritic infection; but as yet it must be considered an open question, whether the exciting cause is not in some cases of a non-specific nature.

It is essentially a disease of childhood, generally occurring between the ages of two and seven years, and in boys more often than in girls.

MORBID ANATOMY.—The affected mucous membrane is at first red and swollen. The epithelium is shed, and a layer of coagulable lymph is then thrown out on the surface, forming a false membrane. The latter varies in thickness from a mere film, to two or three lines or more, and is not very firmly adherent. Gradually, it becomes loosened, and in favourable cases may be partly expelled by coughing, and the parts may gradually return to their normal condition. The inflammation and false membrane extend for a variable distance into the pharynx above and the trachea below, while beyond this point it gradually merges into inflammation of the catarrhal type.

SYMPTOMS.—Signs of an ordinary catarrh, slight fever, and sore throat, often precede the laryngeal symptoms for a few days, but sometimes the onset is more abrupt. The first indication of laryngeal mischief is huskiness of the voice which *gradually* becomes more marked till it is reduced to a whisper. Cough quickly follows, at first hoarse, clanging and paroxysmal, but as the disease progresses the paroxysmal character is lost and it becomes almost or quite toneless. Meanwhile, the breathing becomes increasingly frequent and difficult, and acquires a stridulous quality. Inspiration is at first chiefly affected, but afterwards expiration becomes equally difficult and laborious. The alæ nasi are in active motion, the epigastrium and lower part of the chest sink in instead of expanding at each inspiration, the face and general body-surface become dusky and livid; and the child tosses about clutching at his throat as if to remove some obstacle there situated. Dangerous suffocative attacks are also apt to occur from time to time and chiefly at night. They are due sometimes to accumulations of mucus in the larynx, and sometimes to spasm of the glottis. There is no expectoration at first, but later on viscid mucus with fragments of false membrane may be expelled. The temperature is raised —sometimes considerably; the strength is gradually diminished; the skin is dry and hot at first, but as prostration supervenes it becomes cold and clammy; the pulse is frequent, and ultimately small and feeble; and the glands at the angle of the jaw are often somewhat swollen and tender.

The disease progresses in this fashion for some four or five days, when—in favourable cases—possibly after the expulsion of false membrane, the cough becomes looser and louder, the breathing easier, and recovery sets in slowly. More often the issue is fatal, the respiratory difficulties increase, the paroxysms become more alarming, the skin is cold and clammy, the eyes are dull, the complexion is livid, until death occurs with signs of convulsive asphyxia; or the child may pass into a state of coma due to carbonic acid poisoning. Death is most frequent on the fourth day.

By means of auscultation, information may be gained as to the amount of air entering the lungs. The *breath-sounds are weak in proportion to the obstruction*, though they are often masked by loud laryngeal sounds. Sonorous and mucous râles are usually present, indicating bronchitis. Broncho-pneumonia and pulmonary collapse are also common complications.

It is necessary to distinguish true croup from the less important and less fatal spasmodic laryngitis. The most striking characteristics of true croup are (1) the slow steady progressive increase of symptoms as contrasted with the intermittent progress of false croup; (2) the much greater prostration; (3) the frequent presence of false membrane in the pharynx, and its occasional presence in the sputa; (4) the less acute invasion; (5) the more complete suppression of all tone in the voice and cough; (6) the less marked fever (as a rule); and (7) the greater intensity of stridor, especially during expiration.

Treatment.—All cases of acute laryngeal inflammation require rest in bed, warm clothing, a temperature of 70° F. and *air moistened by steam*.

In *adults* frequent inhalations of steam to which tr. benzoin. co. ʒ j., or succus conii ℈ ij. with sodæ carb. gr. xx., may sometimes be added with advantage. External application of hot sponges, or linseed and mustard poultices to the throat is useful. The skin should be excited to action by warm water or vapour baths, warm drinks, and diaphoretic salines; and the bowels should be well opened. Mackenzie recommends the internal direct application of chloride of zinc (gr. xxx. ad ℥ j.), or the inhalation of the atomised spray of tannin (gr. v. ad ℥ j.), or tincture of steel (♏iij. ad ℥ j.), in obstinate cases.

In acute spasmodic laryngitis of *children*, treatment should be begun *as early as possible.* Give a warm bath immediately, to which mustard (℥ ss. to the gallon) may be added, dry quickly, and put to bed in warm flannel. The air of the sick-room should be kept fresh, and impregnated with steam. Internally ½ drop doses of tinct. aconite (every ¼ hour for 2 hours, afterwards hourly), or a simple saline with vin. ipecac. and paregoric may be advisable at first. If there be much debility, quinine is preferable to the saline draught. Locally a mustard and linseed poultice, or a succession of sponges wrung out in hot water may be applied to the front of the throat. During a paroxysm of dyspnœa, a hot bath, hot sponges to the throat, and ʒ j. doses ipecac. wine every ¼ hour till vomiting occurs, are our chief resources. Warm drinks may be given as required. The diet should be light and nutritious. Tracheotomy is seldom required. As urgent symptoms subside, ammonia, iodide of potash, squills, and senega are indicated; and during convalescence, a general tonic regime with change of air is recommended.

The treatment of true croup is conducted on much the same principles, but the following points are worthy of notice. From the extreme tendency to prostration, a good supply of nourishing food is demanded, especially during the early stages, and stimulants are required from the outset. For the same reason lowering treatment of any kind must be avoided. Emetics are required early, and for this purpose, alum (ʒ j. in honey or syrup every 10 or 15 minutes) is preferable. The best medicine for internal use is chlorate of potash with tinct. of steel. A spray of hydrarg. bichlor. solution (1 in 5000) or carbolic acid solution (1 in 80) to the throat is also of service. Lastly, tracheotomy must not be deferred—as too often happens—until the patient is practically moribund; but should be promptly performed on the failure of the preceding measures to relieve the dyspnœa.

CHRONIC LARYNGITIS.

Chronic laryngitis may occur as a sequel of the acute form; or it may depend upon over-straining of the voice, the inhalation of irritating matter, new growths, syphilis, chronic alcoholism, or the abuse of tobacco.

Whatever be the cause, the *symptoms* are those common to laryngeal mischief of all kinds, as already described. They vary only according to the seat of the lesion, and the physical changes induced by it. The employment of the laryngoscope is therefore essential for a proper diagnosis.

Simple chronic catarrh causes but little discomfort in the larynx. The laryngoscopic changes are those of general or partial congestion and swelling of the mucous membrane. The ventricular bands and epiglottis are most frequently affected. Patches of mucus are met with on the surface here and there.

In "*Clergyman's sore throat*" the racemose glands of the larynx and follicles of the pharynx are enlarged and congested. It either extends from the larynx to the pharynx or *vice versa*. The symptoms are usually milder than in simple catarrh, but often very intractable.

New growths in the larynx may be either malignant or benign. To the former class belong epithelioma, and cancer (very rare); while to the latter belong papillomata, mucous polypi, and fibrous growths. They can only be diagnosed with certainty by means of the laryngoscope. As to the symptoms it may be noted that the amount of alteration in the voice and dyspnœa is liable to sudden variation; that when the growth is above the glottis, expiration is often quite free; and that since obstruction of the passage is the rule, *stridor* is a frequent symptom.

Syphilis.—Chronic laryngitis may occur both in the secondary and tertiary stages of syphilis, but more frequently in the latter. In the former we meet with condylomata, and superficial ulceration; while in the latter we find extensive destructive ulcers; the cicatrisation of which is very apt to be attended with serious constriction of the larynx, and more or less distortion of its parts. Tertiary ulcers are especially prone to attack the epiglottis.

Tubercular laryngitis.—The larynx suffers in some way or other in a majority of cases of phthisis, but more often the affection is of a simple catarrhal nature. A patchy distribution of catarrhal changes is, however, more significant of tubercular origin than when uniformly distributed. Among the more characteristic appearances are ulceration—most frequently situated on the posterior part of the vocal cords—and pale localised swellings which may affect the epiglottis, ary-epiglottic folds, the inter-arytænoid space, or ventricular bands. Pyriform swelling of the ary-epiglottic folds is especially characteristic. These swellings are apt ultimately to break down and ulcerate. Since *necrosis of the cartilages* more often results from laryngeal phthisis than from all other causes put together, it may be mentioned under this head. Necrosis is generally associated with ulceration, the inflammation extends to the perichondrium, under which pus forms and collects, and the cartilages necrose and may even be expectorated in fragments. The prognosis of laryngeal phthisis and its sequelæ is most unfavourable.

GENERAL TREATMENT OF CHRONIC LARYNGITIS.—All varieties urgently require rest for the larynx, a dry air of equable temperature, warm clothing, avoidance of exposure, and removal of any source of irritation. Constitutional remedies are more especially useful in the syphilitic and phthisical varieties, where the usual treatment of those diathetic conditions is indicated. Should deglutition be very painful or difficult, only liquids thickened with cornflour or arrowroot should be given, and in some cases nutrient enemata are of service.

Local remedies are of most benefit. These may be employed in the form of inhalations of vapour or atomised spray, or of liquids applied directly by means of a special brush. The introduction of powders cannot be recommended. For *inhalation*—steam medicated with the oils of pine or juniper, creasote, or carbolic acid is suggested; for *sprays*, alum (gr. x.), tannin (gr. v.), perchloride of iron (gr. ij.), chloride of zinc (gr. ij.-x.), each to the ounce of water; for *direct application*, chloride of zinc is the best for general purposes, but in phthisis—tannin, and in syphilis—nitrate of silver (gr. xx. ad ℥ j.), or sulphate of copper (gr. x. ad ℥ j.) are often more useful. Such applications should as a rule be made daily for the first week, after which less often.

In extreme cases tracheotomy may be required; and certain forms of new growths need removal by operation.

NEUROSES OF THE LARYNX.

Laryngismus Stridulus.

Laryngismus is a disease characterised by spasmodic approximation of the vocal cords, constricting the laryngeal passage, and hence giving rise to dyspnœa and stridor.

The causes of the spasm may be:—(1) *central*, such as hydrocephalus or some organic disease or functional disturbance of the brain; or (2) *peripheral* (*a*) from direct irritation of the recurrent or vagus nerves by enlarged strumous glands in the mediastinum, or (*b*) from indirect irritation at a distance, due to worms, difficult dentition, an over-loaded stomach, &c. It is most common between the ages of six months and two years, and is especially predisposed to by rickets and scrofula.

Symptoms.—The attack begins suddenly in the night without previous warning, with dyspnœa and crowing inspiration which in a few minutes may pass off, and the child after crying with fright soon falls asleep again. In other cases, the severity and duration of the attack is more marked; while in the worst forms of the disease, all respiratory sounds may actually cease for a short time, owing to complete closure of the glottis, and death from asphyxia seems imminent. At this crisis, however, the laryngeal spasm as a rule suddenly ceases, and the attack quickly subsides. Spasmodic flexion of the thumbs and great toes, and sometimes more or less general convulsion are apt to be associated with the laryngeal phenomena. There is no pyrexia. Such attacks are very prone to recur, but with appropriate treatment, the prognosis is usually favourable. Of course, in cases, dependent on incurable organic nervous disease, recovery is hopeless. Death sometimes occurs from asphyxia.

Laryngismus differs from acute laryngitis, by its sudden onset, the absence of fever, the convulsive symptoms with which it is associated, and the complete cessation of all symptoms between the paroxysms.

Treatment.—*During the attack* the child should be placed in a warm bath, and cold water may be dashed on the face. Slapping or rubbing is sometimes recommended. In bad cases inhalation of chloroform may be resorted to.

After the attack the source of the complaint should be thoroughly investigated. If thought to be due to an over-loaded stomach, or the presence of indigestible food, an emetic or purgative is useful. Similarly, lancing the gums, or vermicide remedies may be required. Medicinally, nothing in a general way gives more relief than periodical doses of bromide of potassium. Attention to the diet, the exhibition of tonics, and change of air are necessary to complete the cure. Tracheotomy is rarely, if ever, required.

LARYNGEAL PARALYSIS.

Four varieties are described.

1. BI-LATERAL PARALYSIS OF ADDUCTORS.—Most commonly caused by hysteria. The voice is lost (*aphonia*), but cough-sounds are unaffected. The laryngoscope shows that on attempting to speak the vocal cords either do not move at all, or only approach each other imperfectly. The prognosis is very favourable.

2. UNI-LATERAL PARALYSIS OF ADDUCTORS.—Caused by local injuries, chronic poisoning by lead or arsenic, pressure on the recurrent nerve, or diphtheria. There is dysphonia, and the sound of coughing is always altered, and somewhat toneless. The laryngoscope shows that the affected cord remains at the side of the larynx, and does not approach the middle line on attempted phonation.

3. BI-LATERAL PARALYSIS OF ABDUCTORS.—Generally a result of cerebral disease. Laryngoscopy shows that the cords do not separate on inspiration. The voice is but little affected, but the least exertion brings on inspiratory dyspnœa and stridor. The cough is often croupy.

4. UNI-LATERAL PARALYSIS OF ABDUCTORS may arise from cerebral disease, but more often from pressure on the vagus, or on one recurrent nerve. The symptoms are similar to, but not so severe as when both cords are affected. Laryngoscopy shows that the cord on the affected side is not drawn outwards on inspiration.

The *treatment* is for the most part that of the affection to which the paralysis is due. Paralysis of the adductors due to hysteria may be often cured by faradisation; one pole being placed at the back of the neck, while the other is either introduced into the larynx, or applied to it externally. Paralysis of the abductors may call for tracheotomy.

BRONCHITIS.

Bronchitis is a catarrhal inflammation of the mucous membrane lining the bronchial tubes, which may on the one hand be limited to the trachea and larger bronchi (*simple bronchitis*); or on the other hand, may extend to their ultimate ramifications *(capillary bronchitis)*. With respect to its course, it may either be *acute* or *chronic*.

Acute Bronchitis.

This condition usually arises from "catching cold;" but it may also occur from the introduction of irritating vapours, or solid particles into the bronchi; or as a symptom in many acute specific fevers; or lastly, it may be epidemic as in influenza. Previous attacks, early or advanced age, constitutional debility, and changeable damp climate are common pre-disposing causes.

Morbid Anatomy.—As in all catarrhs, we meet with congestion, swelling and softening of the mucous membrane. In the earlier stages the epithelium is shed, and the secretion is scanty; but later on there is active cellular proliferation and mucous exudation. While in the larger tubes the swollen mucous membrane does not appreciably lessen the calibre, in the capillary tubes it is quite otherwise. Occlusion of the latter readily occurs, the passage of air to and from the air-cells is impeded or prevented, and hence emphysema and pulmonary collapse are common complications.

Again, since the terminal bronchi merge into the air-cells, the catarrhal process is very apt to extend from the former into the latter, and set up catarrhal pneumonia.

SYMPTOMS.—1. *Simple bronchitis* is usually ushered in with chilliness, febrile disturbance and general malaise. A sense of constriction about the chest, and sub-sternal soreness soon follow. The respiration is increased slightly in frequency; cough is uniformly present, is worse after sleep, and often paroxysmal; the expectoration is at first scanty and viscid, but soon becomes more abundant, white, and frothy, and still later, yellowish or muco-purulent. At this stage the cough is looser, less painful, and the expectoration more easy.

2. *Capillary bronchitis* being generally preceded by the milder form just described, its onset is less marked than might otherwise have been expected, but rigors sometimes occur. The patient breathes with difficulty, the complexion is dusky, and the countenance betrays anxiety. The superficial veins are more or less engorged, and the alæ nasi movements are exaggerated. The respiration and pulse are very frequent, the former out of proportion to the latter; the cough is almost perpetual; the sputa are at first frothy, but soon become muco-purulent, and are expelled with considerable difficulty. Sub-sternal pain is not a marked feature, but troublesome pains in the inter-costal muscles, brought on by the cough, are of frequent occurrence. The temperature rises, and the general restlessness becomes extreme. The urine is scanty, high-coloured, and sometimes contains a little albumen.

In fatal cases the prostration increases; the skin becomes more livid, and is cold and clammy; anasarca supervenes; till at last fitful somnolence and muttering delirium usher in coma and death.

The duration varies according to the severity of the case from one to three weeks. Relapse is less common than recurrence. Capillary bronchitis terminates either by (1) death, (2) recovery, or (3) passage into a chronic form.

Physical Signs.—There is no alteration on percussion, but the breath-sounds are coarse and noisy, and are accompanied (or perhaps masked) by sonoro-sibilant and mucous rhonchi, the former of which indicate irregular obstruction of the air-tubes by viscid secretion, while the latter point to the bubbling of air through more liquid secretions. These rhonchi are modified by cough. The milder kind of bronchitis yields no physical indications whatever.

The *diagnosis* of simple bronchitis offers no difficulty. Capillary bronchitis is distinguished from *catarrhal pneumonia* by the greater fever and disturbance of breathing, the dulness on percussion, tubular breathing and irregular distribution of the latter affection. In *acute tuberculosis* the violence and irregularity of the fever, the rapid collapse, and the extreme dyspnœa, out of all proportion to the physical signs, indicate the nature of the case. Care should be taken not to confound cases of primary bronchitis, with specific fevers (*e.g.*, typhoid) in which the catarrh is symptomatic.

As to *prognosis*, early childhood, advanced age, previous organic disease of the lungs or heart, and the occurrence of complications, are especially of unfavourable import.

Treatment.—The patient should be confined within doors, and if necessary to his bed, a warm equable temperature being maintained. It is important to get the skin to act freely by hot air, vapour, or water baths, warm diluent drinks, and diaphoretic salines, combined (where there is great restlessness) with a small dose of Dover's powder at night. Such treatment will if begun promptly, often nip the development of the affection in the bud. Relief is often obtained by steam inhalations, and external application of poultices or turpentine stupes to the chest.

As to medicine, *expectorants* are often of service, but in the febrile stage, only those of a sedative kind should be used, *e.g.*, vin. ipecac. and vin. antimoniale; but later on the more stimulating expectorants are indicated. *Sedatives* are useful to allay the cough, *e.g.*, opium, henbane, conium, and hydrocyanic acid; but where the secretion is abundant they should be used with caution. *Anti-spasmodics*, *e.g.*, ether, chloroform, and tincture of lobelia, may be combined with any of the foregoing. As the acute symptoms subside, tonics are of great service.

The diet should be liberal, nutritious, and supporting. Stimulants are seldom necessary, except in the case of the aged and cachectic. Dry cupping over the chest often gives great relief; but venæsection should only be practised where death is imminent, and there is great venous engorgement.

Chronic Bronchitis.

This is a very common disease, its frequency increasing with age. It is often chronic from the outset, especially when associated with cardiac, renal, or gouty affections; but quite frequently it occurs as the sequel of acute or sub-acute attacks. Chronic lung affections of other kinds, chronic alcoholism, and the introduction of irritating matter into the tubes are also common causes.

MORBID ANATOMY.—The mucous membrane is dark-coloured, thickened, and its vessels are dilated. The bronchial walls gradually become thick and indurated, the tube-cavities may be narrowed or dilated, and usually contain more or less puriform matter, which is sometimes very fœtid.

SYMPTOMS.—The following are the most important varieties of chronic bronchitis:—

1. "*Winter-cough.*"—The cough is violent, most severe in the mornings, the expectoration being scanty and difficult to expel; or abundant and easy, consisting of yellowish-green matter, which runs together, is only slightly frothy, and sometimes very fœtid. There is not much pain or soreness except after violent coughing; neither is there much dyspnœa except after exertion. The general health suffers, but there is no fever. At first the symptoms are comparatively slight, and are confined to the winter months, disappearing in the summer. The attacks tend to become more severe, and to last longer, till the patient is never quite free. Among the aged there is great risk of an acute attack supervening upon the chronic form.

2. *Dry catarrh* is especially characterised by very scanty expectoration. The cough is violent and paroxysmal, the breathing is oppressed, and dyspnœa is sometimes extreme. This form is most common in gouty subjects.

3. *Bronchorrhœa* generally occurs in old people, and is characterised by copious expectoration of a thin watery fluid, but sometimes ropy and glutinous, and occasionally fœtid. The cough and dyspnœa are marked. This variety is especially apt to be associated with bronchial dilatation (bronchiectasis).

The *physical signs* of chronic bronchitis are in the main similar to those of the acute form.

Chronic bronchitis becomes a serious affection from its leading so frequently to emphysema, collapse of lung, and catarrhal pneumonia, and thus after it has become well developed is only curable in a very restricted sense.

The *treatment* depends very much on the age and constitution of the patient. Expectorants are first indicated, followed at a later period by tonics (of which the astringent preparations of iron are perhaps the best), and cod-liver oil. In gouty bronchitis—iodide of potassium with colchicum; in bronchorrhœa—turpentine, the balsams of tolu and Peru, or ammoniacum; in winter-cough—tar (in pill containing gr. ij.) every four hours, or inhalation of a spray of pure or diluted vin. ipecac. is recommended. Inhalations of steam impregnated with oils of pine, juniper, or turpentine, are sometimes useful.

The external application of poultices, sinapisms or stupes, and occasional hot air or vapour baths often give great relief.

BRONCHIAL DILATATION.

Dilatation of the bronchi, or bronchiectasis, though a purely secondary affection, deserves separate mention both on diagnostic and prognostic grounds. The dilatation may affect a considerable length of one or more bronchial tubes *(general dilatation)*, or be limited to particular parts *(saccular dilatation)*. The bronchi of the middle and lower lobes of the lung are more frequently affected than those of the upper lobe. The dilatations usually contain pus or muco-pus, more or less inspissated, and sometimes horribly fœtid. The mucous lining may be unaltered, but more often it is either congested and swollen, superficially ulcerated, or atrophied. The pulmonary tissue around is, as a rule, condensed by collapse or fibroid change. Such dilatations are primarily due to structural alteration of the walls of the bronchi, by which their resistant power is diminished, and thus they give way under the influences of repeated coughing, and fibroid shrinkage of lung.

The *diagnosis* of bronchial dilatation rests chiefly upon the history of severe or long-continued antecedent bronchitis, the violent paroxysmal character of the cough, the abundance and not infrequent fœtor of the expectoration, the physical signs of one or more cavities (see Phthisis) in the middle or lower part of the lungs, the slight amount or complete absence of febrile disturbance, and the very slow progress of the disease. Hæmoptysis is not an unusual occurrence at some period or other in the course of the case. Phthisis and pulmonary gangrene are the diseases with which bronchial dilatation is most apt to be confounded. When once dilatation has occurred it is probably permanent. The *treatment* resembles that already described for chronic bronchitis.

SUB-SECTION III.

DISEASES OF THE LUNGS.

PULMONARY CONGESTION AND ŒDEMA.

BOTH on pathological and clinical grounds, congestion and œdema of the lungs should be considered together. Congestion of the lungs may be either *active*, *mechanical*, or *passive*. *Active* congestion may result from (*a*) the breathing of very hot or cold air, or irritating matter; (*b*) circulatory obstruction in another part of the same lung, or in that of the opposite side; (*c*) inflammation, of which it forms the first stage; (*d*) suction-action on the internal surfaces of the air-sacs from inspiratory obstruction. *Mechanical* congestion is a common result of valvular disease of the heart. *Passive* congestion depends upon enfeeblement of the heart's action, and is a common complication of fevers and other conditions characterised by great debility and prostration. Under the influence of gravitation, it especially affects the most dependent parts of the lung *(hypostatic congestion)*. When congestion is severe or long-continued, other changes are apt to follow, *e.g.*, pneumonia, hæmorrhage, and œdema. Œdema of the lungs not only occurs as the result of congestion, but also as part and parcel of general dropsy.

MORBID ANATOMY. — *Congested lung* is denser and heavier than normal, has a dark-red colour on section, and blood-stained frothy serum flows freely from the cut surfaces on pressure. It crepitates imperfectly on pressure, but floats in water.

When *œdema* is present, the lung-tissue is swollen, does not collapse when the chest is opened, and pits on pressure unless the œdema be quite recent. It is pale or dark-red, according as congestion is absent or present, and large quantities of watery serum exude on section.

SYMPTOMS.—These vary according to the severity and duration of the congestion, and the amount of œdema. Cough, dyspnœa, more or less oppression at the chest, together with duskiness or lividity of the cutaneous surface, are the chief symptoms. The expectoration is scanty in the active form, more abundant in mechanical congestion, while it is profuse and watery in œdema. Hæmoptysis may also occur.

Physical examination reveals nothing of importance in simple congestion; but when œdema is present the breath-sounds are weak and somewhat harsh, and are accompanied with thin bubbling râles; the percussion-note also is often diminished. When œdema of the lung occurs as a part of general dropsy, *both bases* are affected, though unequally so, if the patient lie habitually on one side.

The *prognosis* and *treatment* necessarily vary according to the condition to which the congestion is due.

HÆMORRHAGES OF THE LUNG.

Under this head are included (1) hæmorrhage into the tubular air-passages, (2) hæmorrhagic infarction, (3) pulmonary apoplexy. 1. *Hæmorrhage into the tubular air-passages* may arise from *congestion* caused by the inhalation of irritating vapours, paroxysmal cough, valvular disease of the heart, or suppressed menstruation (?); from catarrhal, phthisical, syphilitic, or cancerous *ulceration;* from perforative communication with an *aortic aneurism;* and lastly from disease of the lungs, notably *phthisis* and *cancer.* It should also be remembered that unusual fragility of the vessels, or the hæmorrhagic diathesis may account for some few cases, otherwise inexplicable. Excepting certain rare cases of syphilitic or cancerous ulceration, and perforative communication with an aneurism, the seat of hæmorrhage is almost invariably in the small terminal bronchi.

2. *Hæmorrhagic infarction* is generally regarded as the result of embolic plugging of a branch of the pulmonary artery by a fragment of clot from the right heart or a systemic vein; but, without denying this origin in a certain number of cases, the opinion is gaining ground that

more often it is due to simple hæmorrhage from mechanical congestion, and has nothing to do with embolism (p. 13). A pulmonary infarct or circumscribed hæmorrhage forms a more or less wedge-shaped nodule or mass, dark-red from infiltration with blood, of firm consistence, with every variety of size. Necrosis rarely occurs. As a rule, the blood is absorbed, and the part either recovers or shrinks into a brown indurated nodule. When of an infective nature, a metastatic abscess follows.

3. *Pulmonary apoplexy*, or diffuse hæmorrhage from rupture of a pulmonary vessel, may occur from extreme congestion due to heart disease, from disease of arterial walls or from external injury. The lung-tissue is lacerated and an irregular potential cavity is formed, which becomes infiltrated with clotted blood.

SYMPTOMS.—*Hæmoptysis* is the characteristic symptom of pulmonary hæmorrhage. It varies from a few streaks in the sputa, to large quantities of pure blood. When the amount is small it is expelled by spitting; when larger it escapes in gulps, or with efforts scarcely distinguishable from vomiting. The blood is usually bright red, alkaline, and frothy; but when its discharge is delayed it may be dark coloured; and in large quantities it may be non-aerated. Attacks of hæmoptysis are prone to recur; and for some days after each, the sputa are always more or less tinged. When the hæmorrhage depends on some disease of the lungs, or the blood is sucked back into the air-tubes, moist râles may be heard in certain parts of the chest. A single attack is rarely fatal, and the ultimate prognosis depends almost entirely on the cause of the bleeding. A careful inspection and the dark colour of the blood will usually suffice to distinguish bleeding from the nose or mouth; while laryngoscopic examination, and the presence of laryngeal symptoms will enable us to detect hæmorrhage from this source. Hæmoptysis will be contrasted with hæmatemesis when describing the latter. The special points to be noted are the ejection by spitting, the bright-red colour, the alkaline reaction, the frothy appearance, the tinging of the sputa afterwards, and the usual co-existence of diseases of the lungs, heart, or mediastinum.

Assuming the ability to exclude bleeding from the nose, mouth, stomach, and larynx, it is not always easy to refer hæmoptysis to its exact source. Careful examination of the lungs, heart, and mediastinum is of the first importance, and the results thus obtained, must be considered together with the age of the patient, the history of the case, and the general condition. Hæmoptysis is more often associated with phthisis than any other affection, and not infrequently constitutes the first obvious indication of this disease ; though on enquiry, it will almost always be found to have been preceded by progressive loss of flesh. Hæmoptysis and dyspnœa suddenly occurring in connection with valvular disease of the heart, or thrombosis of a peripheral vein, point to pulmonary infarction and apoplexy. In these cases an area of consolidation may sometimes be detected, which corresponds to the seat of hæmorrhage. The diagnosis of the rarer forms of hæmoptysis caused by cancer, aneurism, etc., depends entirely on the general evidence of the existence of these conditions.

The general principles of *treatment* to be observed are—rest in bed in the recumbent posture in a cool room, and the administration of hæmostatics, such as ergot (in full doses), gallic acid, acetate of lead, dilute sulphuric acid, or turpentine. In some cases the addition of an opiate is desirable to calm the nervous system and to allay the cough. Ice may be sucked, or applied (with caution) to the chest-wall. The bowels should act freely, especially in plethoric habits. Dry cupping over the chest, and hot stimulating foot-baths are often of service.

ACUTE CROUPOUS PNEUMONIA.

ACUTE CROUPOUS PNEUMONIA is an inflammation of the lung which is characterised by a fibrinous exudation into the air-cells. With reference to its causation, exact knowledge is wanting. There are many reasons for regarding pneumonia as the local manifestation of some more general disease, which may perhaps be of a specific nature. In most cases, exposure to cold appears to act as an exciting cause, especially when the body is heated. Pneumonia also often supervenes as a secondary affection in other diseases.

MORBID ANATOMY.—Three stages are generally described.

(1) The first *stage of engorgement* or congestion is similar to that already described under the head of active congestion of the lung. (2) In the *stage of exudation or red hepatisation* the inflamed lung is of dark-red colour, enlarged, solid, inelastic, non-crepitant, and heavy. It breaks down readily on pressure, and sinks in water. On *section*, the surface is dark-red, mottled with grey, and presents a peculiar *granular* appearance which is less marked in children and old people than in adults. Microscopically, the vesicles and terminal bronchioles are filled with a fibrinous exudation, enclosing both red and white blood-cells. Up to this point there is little or no alteration in the alveolar walls. (3) The *stage of grey hepatisation* is mainly characterised by cell multiplication and degeneration. The colour gradually changes

from dark-red to grey or yellowish-white. The weight, density, and friability of the lung are increased, and the granular appearance on section is less marked. Microscopically, excessive multiplication of the cellular elements in the exudation and alveolar walls is now met with, and these cells rapidly tend to become granular and undergo fatty degeneration.

Arrived at this stage the process terminates in one of four ways: (*a*) liquefaction of the exudation, and removal by absorption and expectoration, (*b*) caseous degeneration and phthisis, (*c*) gangrene, (*d*) the formation of an abscess.

The *lower lobe of one lung* is generally affected, but the inflammation may involve the whole of one lung, or both. Sometimes it is confined to the *upper lobe*. The rest of the lung is congested and œdematous. An exudation of lymph is commonly found on the pleura covering the affected part.

SYMPTOMS.—In the primary form of pneumonia, the attack generally sets in abruptly with a single severe and prolonged rigor, during which the temperature rapidly rises to a high grade, followed by the usual febrile symptoms. In children the chill is often replaced by severe vomiting or convulsions. Three important local symptoms now occur. (1) *Pain* in the mammary region of the affected side, which is sharp, stabbing, and aggravated by movement, respiratory or otherwise, and also by pressure. (2) *Dyspnœa* which is characterised by extremely frequent shallow breathing, and is not of the suffocative kind met with in bronchitis. It ranges from thirty to sixty per minute and it is due partly to the fever, partly to mal-aeration of the blood, and partly to the pain. The alæ nasi are in active motion. (3) *Cough*, which is frequent, hacking, and restrained on account of the pain accompanying it. It is dry at first, but by the second or the third day it is attended by the characteristic *expectoration of viscid, tenacious, rust-coloured material*, consisting of albumen and mucus mixed with blood and innumerable cells of various kinds. Expectoration does not occur in young children, and is often absent in secondary pneumonia, and in pneumonia of the aged.

General Symptoms.—The *fever* attains its maximum on the second or third day usually ranging from 103° to 105°, and excepting the small daily variations, this temperature is maintained until the *crisis*. The *skin* is excessively hot, dry and pungent. The *pulse* is frequent, from 80 to 120 per minute, usually large and full at the outset; but it afterwards becomes small and weak, partly as the result of imperfect filling of the arteries from obstruction to the pulmonary circulation, and partly through cardiac feebleness. The *cheeks* are flushed, and *herpes* is often present on the lips, which are dusky. Prostration, feebleness, headache, sleeplessness and slight nocturnal delirium, are common *nervous symptoms*. The *tongue* is coated and dry; *thirst* is increased; the *appetite* is lost, and the *bowels* are usually costive. The *urine* is of the febrile type; chlorides are deficient or absent, and it may contain a little albumen.

In severe cases the symptoms often assume an *adynamic* or *malignant* type, marked by grave nervous disturbance, delirium, coma, convulsions, &c., especially in those who are enfeebled by alcoholic excesses, previous disease, or old age.

The symptoms (as described) increase up to the fifth, sixth, or seventh day, when in favourable cases they quickly subside, the temperature rapidly falls to normal, constituting what is called a *defervescence by crisis*. The cough and dyspnœa abate, the sputa become more abundant, of a yellowish colour and muco-purulent.

When the disease ends fatally, death usually occurs at the end of the first, or beginning of the second week, and results from cardiac failure, and respiratory embarrassment. More rarely, it occurs from collapse after the crisis, or from complications.

Physical Signs.—In the *stage of engorgement* nothing is noted beyond *fine crepitation* over the affected part on deep inspiration. In the *stage of red hepatisation*, the movements of the chest are impaired, there is dulness on percussion, the breathing is tubular or bronchial, and the vocal fremitus is increased over the hepatised area. Similar signs characterise the stage of *grey hepatisation*, with the addition of *redux crepitation*, which is moister and of a coarser quality than the crepitation of the first stage. The physical signs persist for a considerable, but very variable, time after the crisis.

Pleurisy, pericarditis, and bronchitis sometimes occur as complications; while—as already mentioned—abscess, gangrene, and pneumonic phthisis constitute occasional sequelæ.

With regard to *diagnosis*, it must not be forgotten that when of secondary origin, or when occurring among the aged, pneumonia is very apt to run a *latent* course. In drunkards, and young children too, the disease is sometimes masked by prominent nervous symptoms. The extreme frequency of the breathing *out of all proportion* to the pulse, and the very sudden onset, are also signs of great value.

As to *prognosis*—old age, infancy, drunken habits, the affection of both lungs, and extreme nervous disturbance are of unfavourable import. Secondary pneumonia (*cæteris paribus*) is more serious than primary pneumonia.

Treatment.—Since little or nothing can be done either to subdue the inflammation, or to shorten the course of the disease, the chief objects of treatment are to sustain the strength of the patient, and relieve symptoms as they arise. Rest in bed is imperative, in a well ventilated room, at an equable temperature. The diet should consist of milk, eggs, beef-essence, &c., and should be given in small quantities at short intervals. Alcohol is always necessary among the aged, also in most cases of secondary pneumonia, and whenever there are evidences of progressive debility and cardiac failure. When indicated, it should be given freely. In the early stage, unless contra-indicated by undue depression, small and frequent doses of aconite and antimonial wine are of great service. Full doses of quinine at short intervals have also been recommended, as tending to check the development of the disorder. Later on, the medicinal indication depends upon circumstances. In simple cases, nothing may be needed beyond some simple saline expectorant. But whenever the age, previous habits and present condition of the patient make it probable that the disease will assume an asthenic type, tonic remedies such as quinine, ammonia and bark, incombination with digitalis, or nux vomica, are urgently required. Symptoms of asthenia, should not be waited for, but foreseen, if possible.

Pain in the side, sleeplessness, delirium, and high fever often call for special treatment. Hot fomentations, sinapisms, stupes, or a few leeches may be employed for the relief of pain. For sleeplessness, tepid sponging followed by a draught of bromide of potash and henbane in camphor water is recommended. If this fails opium may be given, but chloral is most undesirable. Delirium, which is so frequent among habitual topers, may best be met by tepid sheet-packing, ice to the head, and in bad cases by morphia injections. High fever calls for the employment of cold, in the form of baths, sheet-packing, or sponging, together with full doses of quinine.

CATARRHAL PNEUMONIA.

Catarrhal, or lobular pneumonia, is *always secondary to bronchitis;* either by the *direct* extension of the catarrhal inflammation into the alveoli, or *indirectly* by catarrhal changes occurring in collapsed lobules. Hence its causes are those of bronchitis; and for similar reasons, it is of frequent occurrence in the course of measles and whooping-cough. As an *acute* affection it is more common in children; but the *chronic* form is more common in adults, and usually results from chronic bronchitis. The acute form only will now be described, the chronic form being referred to under the head of phthisis.

Morbid Anatomy.—Acute catarrhal pneumonia is almost always at first limited to single lobules, scattered throughout the lung, especially in the lower lobe. Nodules of consolidation are thus formed, which are non-crepitant on pressure, friable, and vary in size according as they remain separate or merge with others. The surface on *section* is smooth and of a greyish-red colour. Yellowish or purulent-looking non-frothy fluid can be expressed from the cut ends of the small bronchi, which are often dilated. Under the microscope, *the vessels and terminal bronchi are observed to be stuffed with cells in various stages of fatty degeneration.* In favourable cases these products liquefy, and are then absorbed or expelled; but occasionally they form caseous masses, and lead to "consumption" of lung-tissue. The above description applies in the main to catarrhal pneumonia, whether it results directly from bronchitis, or from "collapse."

Symptoms and Course.—Since acute catarrhal pneumonia is invariably a secondary affection, it will suffice to point out those features which characterise its invasion. Its onset may be *abrupt*, as in measles, or *gradual*, when it supervenes on collapse, as is usual in whooping-cough.

The more important signs are—(1) a more or less sudden accession of fever, the temperature rising to 103° or 105°, together with a corresponding rise in the frequency of the pulse; (2) increased dyspnœa; (3) a change in the character of the cough, which becomes short, hacking, and painful; (4) diminished expectoration (if present). At the same time the child is very restless, and in bad cases is apt to fall into a state of coma.

Physical examination in the more trivial forms gives no characteristic results; but where the nodules are large and numerous, there is dulness on percussion, bronchial breathing, moist crackling, and bronchophony.

The progress is sometimes rapid, and death may ensue in a few days. More often, however, it is prolonged, and the defervescence is gradual. Occasionally it terminates in acute phthisis.

Treatment.—No lowering treatment is admissible. Internally, quinine in full doses generally answers better than anything else; ammonia and bark, with or without soothing expectorants, is also a useful combination. Good nourishment is essential, and alcohol is often requisite. Brandy is the best form, and may be given to children in doses of ʒ ss.-ij. every two or three hours. Externally, hot and moist applications are recommended, as in croupous pneumonia. During convalescence tonics are indicated.

INTERSTITIAL PNEUMONIA—FIBROID PHTHISIS.

This disease is essentially *chronic*, and is characterised by excessive *over-growth of the pulmonary connective tissue*, and is usually associated with *dilated bronchi*. When first formed, it is soft and rich in cells, but afterwards is converted into dense contracting fibrous tissue, which ultimately more or less completely replaces the proper tissue of the lung.

In the vast majority of cases it is clearly a *secondary* affection, supervening upon chronic catarrhal pneumonia, bronchitis (or according to some authorities) upon certain dry pleurisies. Whether it is ever *primary* is still doubtful. The dilatation of the bronchi is usually *secondary* to the induration, and is mainly due to the shrinking of the lung, which leads to distension of the tubes, by the pressure of the air within them, the effects of which are probably enhanced by coughing and inflammation. In some of the most marked cases the change is often confined to one lung.

Morbid Anatomy.—The *lung* is hard, dense, and contracted. Its surface on section is smooth, of a marbled grey colour, and is traversed by white fibrous bands. The vesicular tissue is destroyed. Here and there caseous masses may often be seen. The *bronchi* present fusiform or saccular dilatations of variable size, the interior surfaces of which are irregular or ulcerated, and often filled with fœtid secretion (*bronchiectasis*). The *pleura* is thickened and adherent.

Symptoms.—The chief *local* symptoms are—(1) *cough*, which is incessant, and often paroxysmal, coming on especially in the early morning; (2) *dyspnœa* which is more marked after exertion; and (3) *expectoration*, the amount and character of which vary according to the condition of the bronchi. When the bronchi are dilated, the expectoration is abundant, puriform, and very fœtid. Slight hæmoptysis is not uncommon. There is gradual loss of flesh and strength, and sometimes night-sweating is complained of. There may be a trifling amount of irregular fever, but, in the absence of bronchitis, there is more often none at all. Albuminuria, from chronic renal disease, is common in the later stages.

Physical signs.—(1) Retraction of the affected side with deficient movement; (2) tubular or wooden dulness on percussion; (3) the breath sounds are bronchial and cavernous; (4) moist sounds of various kinds, often of a metallic quality; (5) increased vocal fremitus; (6) bronchophonic or pectoriloquous vocal resonance; and (7) frequent displacement of the heart's apex towards the affected side. The right heart is often dilated.

The *treatment* is the same as that of phthisis, and will be considered under that head.

PHTHISIS—PULMONARY CONSUMPTION.

The old definition of phthisis was a clinical one based upon the more or less rapid emaciation, and exceptional recovery which characterised certain diseases of the lungs, due—according to Laennec—to the deposit in those organs of a new growth, which he called tubercle. But it was afterwards ascertained, that what had been called yellow tubercles, were to a large extent merely the products of ordinary inflammation, which had undergone caseous degeneration. This in time led to the view, that the vast majority of cases of pulmonary phthisis, where the exclusive result of inflammatory (pneumonic) changes of a low type, and that grey tubercles, if present, were of secondary and incidental origin. At the same time, it was admitted that at least in some cases, the tubercular formation constituted the primary change, and that the pneumonic condition was secondary.

In the light of recent knowledge, these views require still further modification. More careful observation has shown that the presence of true tubercle is almost, if not quite an invariable feature of pulmonary phthisis in all stages; and the bacilli, which are pathognomonic of tuberculosis, are always found in the sputa of phthisical patients at one time or other in the course of the disease. Admitting these facts, pulmonary phthisis must be regarded as a *localised tuberculosis*. In other words, it is a *specific* disease, which is primarily due to the introduction and development of tubercular bacilli within the lungs. The specific cause, however, cannot take effect under ordinary circumstances, in the absence of either an inherited or acquired predisposition on the part of the individual exposed to its influence. The predisposing causes of phthisis are identical with those of tuberculosis in general, and the reader is therefore referred to the chapter on this subject (p. 112).

Speaking generally, the changes which are met with in phthisical lungs are the results of inflammation, characterised by two special features:—(1) The specific nature of the circumstances under which it arises; and (2) the destruction and necrosis of lung-tissue by which it is invariably followed. The inflammatory process is sometimes acute, and sometimes chronic. In the former, the destruction is rapid, and far-reaching; while in the latter, an over-growth of interstitial and peribronchial connective-tissue accompanies the destructive process, and is well marked in direct proportion to the chronicity of the disease. The differences are not quite easy to explain, but they probably have largely to do with the amount of the virus introduced, the general constitution of the patient, and the condition of the lungs at the time of infection. Acute miliary tuberculosis of the lung, which only forms a part of more widely spread tuberculosis, is not now under discussion, as it does not come within the category of phthisis.

Acute Phthisis.

In acute phthisis the introduction of the tubercular virus sets up an acute capillary bronchitis, which by extension into the alveoli, quickly leads to acute catarrhal pneumonia. The special feature of the process consists in the rapid implication, with suppuration and destruction, of the lung-tissue. From the rapidity with which the tubercles degenerate and break down, together with the destruction of the tissue around, few tubercles, or none at all, may be found post-mortem.

Symptoms.—The disease is ushered in with chills, and fever of the hectic type. Cough, dyspnœa on slight exertion, profuse expectoration, sometimes tinged with blood, soon follow. The pulse is frequent and feeble, the tongue is red or furred, with anorexia and frequent diarrhœa. There is great exhaustion, profuse sweating, rapid emaciation, and restlessness passing on to delirium. It is almost always fatal.

Dulness on percussion in proportion to the amount of consolidation, bronchial breathing, copious and widely diffused moist or metallic crackling, increased vocal resonance and fremitus, constitute the chief physical signs.

Treatment.—Dr. M'Call Anderson reports cases which he has treated successfully; and recommends rest in bed, liberal supply of fluid nutritious food and stimulants, frequent application of iced-cloths to the chest to subdue the fever (to be at once removed if there is any tendency to collapse), hypodermic injections of atropine to check the sweating, and pills or powders containing quinine gr. j., pulv. digitalis gr. ½-j., and opium gr. ¼-j., every four hours.

Chronic Phthisis.

Anatomical Appearances.—The disease as a rule begins, and is hence most advanced, at the apices of the lungs; and extends, though to an unequal extent on the two sides, from above downwards. The process is usually described as occurring in three stages, viz., *consolidation*, *softening*, and *excavation*. If, however, we bear in mind that it is often impossible to determine at what stage a case has arrived, and that different stages are met with even in the same lung, it follows that such a classification must be of very limited practical importance.

Ignoring for the moment any bronchitic or pneumonic mischief which may precede the phthisical process, and on which the latter is so apt to supervene, the disease begins by the formation of tubercles in the finer bronchi, in association with catarrhal inflammation. Thence the inflammation extends (1) to the alveoli; and (2) to the peri-bronchial connective tissue, and by slow degrees to the interstitial tissue generally. The alveoli become crammed with epithelioid cells; and their walls infiltrated with small round cells (catarrhal pneumonia). Of the nodules of consolidation thus formed, some undergo caseous degeneration; which, beginning in the centre of each, extends peripherally, till the whole is converted into a structureless cheesy mass. In some cases, the caseous mass becomes encapsuled in course of time with fibrous tissue, and perhaps more or less calcified; and the process may thus be temporarily or permanently arrested. More often it liquefies, and cavities are formed, which are at first small and round, but afterwards become large and irregular by coalescence with others. Sooner or later they open into one or more bronchi, and their contents are discharged by expectoration. Their inner surfaces, in the first instance rough and ill-defined, now become smooth and well marked.

Accompanying these changes, as already mentioned, there is an *increased formation of the pulmonary connective tissue*, which is attended with induration and subsequent shrinking of lung tissue. More or less bronchitis, bronchial dilatation, and pleuritic adhesion, are constant accompaniments of phthisical inflammation. Crops of fresh tubercle are constantly appearing, which are variously distributed throughout the lung. The small *pulmonary vessels* also often exhibit inflammatory or tubercular changes in their walls, causing coagulation of their contents. Sometimes they are the seat of *aneurismal dilatations*, which may burst and thus lead to hæmorrhage.

SYMPTOMS.—The onset is usually gradual, and marked by one or more of the following conditions, viz., progressive debility and loss of flesh, chronic dyspepsia, diarrhœa, alterations in the voice (*laryngeal phthisis*), or amenorrhœa. Sometimes profuse hæmoptysis is the first symptom which attracts attention. A precursory catarrh is of common occurrence.

In a general description we may distinguish local and general symptoms.

Local.—*Pain* in the side and about the scapular region is generally complained of, but is rarely severe. *Dyspnœa* is comparatively slight in the absence of exertion, and if marked, is generally the result of high fever or some complication. *Cough* is a constant symptom, but varies greatly both in its character and severity. Sometimes it is paroxysmal and attended with vomiting. *Expectoration* may at first be mucoid and frothy, but it quickly becomes opaque and semi-purulent, and often exhibits a peculiar *striated* appearance. When cavities have formed, the sputa are often compact and globular (nummulated). Microscopic examination reveals *tubercular bacilli*, epithelium, pus, and (sometimes) blood-cells; a large amount of granular matter, and *fragments of pulmonary elastic tissue*. *Hæmoptysis* occurs at some time or other in the vast majority of cases. It varies from a few streaks to a large quantity of pure blood; and frequently, but not necessarily, follows a fit of coughing.

General.—*Fever* is a constant feature of all cases of phthisis *in progress*. It varies much in its intensity and its type, and runs a very irregular course. As a rule, the intensity is in direct proportion to the rapidity of the consumptive process, and it conforms to the *hectic* type. Profuse *night-sweating* is a constant drain upon the system, and contributes largely to the general enfeeblement. *Emaciation* is noticeable from the first, and its rate of progress constitutes the best criterion of the general progress of the disease. The skin is pale, and often dry and scaly, and there is sometimes a little œdema about the ankles. The *finger-ends* are often *bulbous*, and the *nails incurved*. *Digestive disturbance* is indicated by a red or a slightly furred tongue, nausea and vomiting, bad smelling breath, loss of appetite with a special dislike for fats, and irregular attacks of diarrhœa. The *urine* is of the febrile type.

The chief *complications* are pneumothorax from perforation of the pleura, tubercular affections elsewhere (especially of the intestines, due to inoculation from swallowed sputa) albuminoid diseases of various organs, fatty degeneration of the liver, fistula in ano, thrombosis of femoral veins, &c. Laryngeal tuberculosis is not infrequently associated with pulmonary phthisis.

The *duration and course* of phthisis vary so much as to defy description. Sometimes, when taken in hand early, the disease may be arrested; but much more often it is ultimately fatal, and death occurs from asthenia, or from hæmoptysis, or from some complication.

Physical Signs.—1. The *thorax* is usually small and ill-developed, conforming either to the *alar* or *flattened* type. As the case advances, the supra- and infra-clavicular regions tend to become depressed from fibroid shrinking of the subjacent apices of the lungs; and the movements of these parts are diminished. As a result of *consolidation* the vocal fremitus is increased; and there is dulness on percussion over one or both apices of the lungs, extending downwards to an unequal extent on the two sides. In the absence of concurrent pneumonia, the percussion note may not be materially affected just at first. The breath-sounds, generally weak and jerky in the first instance, become bronchial or blowing. Dry or moist crackling râles, occurring for the most part during inspiration, indicate commencing softening. The vocal resonance is bronchophonic. The heart is sometimes slightly displaced, and its impulse diffused, owing to shrinking on the part of the lungs; while the sounds are heard with unusual distinctness under the clavicles. A systolic bruit is also often heard over the subclavian artery in the same region.

The following are the chief signs of a *cavity*:—a tubular or cracked-pot sound on percussion, especially when the mouth is open; cavernous or amphoric breath-sounds; large, hollow, metallic or ringing râles, or metallic tinkling; whispering pectoriloquy; while the heart sounds are generally intensified, and sometimes acquire a hollow quality by transmission through the cavity.

Certain clinical varieties of chronic phthisis may be briefly noticed. 1. In so-called *catarrhal phthisis*, there is always a precursory bronchial catarrh, lasting sometimes for many years. Its onset is very insidious, and its progress slow. This form is common among the young, after measles or whooping cough, or after any form of chronic bronchitis attended with constitutional debility. 2. The term *fibroid phthisis* is applied to those cases in which interstitial fibroid overgrowth is a marked feature. They run a very chronic course. This form is rarely, if ever, seen except in adults, with whom it may occur as a sequel of bronchitis from long-continued inhalation of dust, &c., or in a lung imperfectly recovered from the effects of pleurisy. 3. In purely *tubercular phthisis*, the onset is very insidious. For a considerable time continued fever, progressive emaciation and weakness, and hacking cough, may be the only symptoms; while physical signs are inconspicuous, or wanting altogether. In such cases, there is a marked tendency to secondary tubercular mischief in other organs. 4. Cases in which attacks of profuse hæmoptysis are of frequent occurrence, are sometimes distinguished by the term of *hæmorrhagic phthisis.*

TREATMENT.—Only a brief outline of the main principles and indications for treatment can be given here.

Fresh pure air is all-important, phthisical patients bearing free ventilation well; while moderate exercise, with careful avoidance of fatigue, light yet warm clothing, cold or tepid bathing, and abstention from close unhealthy occupations are no less necessary.

The *diet* should be liberal and nutritious, including some easily assimilable fats. *Alcohol*, in so far as it is required to aid digestion, is often beneficial.

Bitter tonics, mineral acids, quinine, iron, and especially cod-liver oil, are of great service. The last must be given in small doses (ʒ ss.-j.) at first, and increased afterwards if necessary. It may be taken in milk, or orange wine, preferably after meals. Foster recommends the addition of ether (♏x. or xv. to the drachm of oil). Dobell gives fat in the form of pancreatic emulsions. Tonics, however, and especially cod-liver oil, are of no use while the stomach and bowels are out of order. These require close attention. When the tongue is furred, and digestion feeble, with irregular action of the bowels, an alterative pill should be given occasionally, with a mineral acid and some light vegetable bitter. When the tongue is raw, and the stomach irritable, bismuth is useful, combined with an alkaline bicarbonate, and hydrocyanic acid; and light fluid food.

Sweating can best be checked by subcutaneous injection of atropine (gr. $\frac{1}{120}$-$\frac{1}{60}$). The administration of small doses of Dover's powder or oxide of zinc (gr. iij. in a pill) the last thing at night, strychnia, sulphuric acid, or tepid spongings of the surface with acidulated water may be employed for the same purpose. *Chronic discharges*, such as diarrhœa or leucorrhœa, should be stopped as far as possible. For the former, bismuth with Dover's powder is a useful combination.

The treatment of hæmoptysis has been dealt with elsewhere. *Cough mixtures* containing opiates should be avoided as far as possible, for many reasons; but if employed, the addition of some preparation of belladonna is desirable, to neutralise the constipating effect of the opium. Among the best remedies for this purpose may be mentioned, the balsams of tolu or Peru, hydrocyanic acid, hydrobromic acid with spirits of chloroform (Fothergill), and inhalations of steam medicated with conium or hyoscyamus. *Counter-irritant applications* to the chest, such as iodine, sinapisms, and flying blisters often give great relief to the pain and to the cough.

The question of change of *climate* frequently arises. Our guiding principles in the selection of a locality should be pure air, equable temperature, good drainage, pleasant surroundings, dryness rather than moisture of the air, bracing rather than relaxing climate—preferably in the neighbourhood of the sea, or at high altitudes if inland; in short, a climate which admits of the patient being as much in pure open air as possible.

EMPHYSEMA.

The term *emphysema* is applied to two different conditions; (*a*) to air in the connective tissue of the lung, or *interlobular emphysema;* (*b*) to a morbid enlargement of the air-vesicles, or *vesicular emphysema.*

Interlobular Emphysema is a condition of little clinical importance. Occasionally as the result of a wound from without, or of a violent *expiratory* effort, one or more air-vesicles are ruptured, and the air escapes into the neighbouring connective tissue, forming on the surface of the lung rows of little bead-like bubbles corresponding with the boundary lines of lobules. They can be removed by pressure. Air thus extravasated may find its way into the mediastinum, or the areolar tissue of the neck; and it is only under these circumstances that interlobular emphysema can be diagnosed during life.

Vesicular Emphysema.—In accounting for the causation of vesicular emphysema, it must be borne in mind that no permanent enlargement of the air-cells of the lung can take place without involving a loss of elasticity on the part of their walls. It is exceedingly doubtful whether any increased tension of elastic tissue which is likely to occur could destroy its elasticity, or impair it to any very serious extent, so long as the tissue remained structurally sound and healthy. But assuming a disordered and impaired nutrition of the pulmonary elastic tissue, the result is very different. A comparatively moderate amount of tension if frequently repeated or long-continued, would then suffice to bring about permanent lengthening of the elastic fibres, and enlargement of the included air-sacs.

It is probable that in all cases of emphysema, the *primary* change consists of an impairment in the nutrition of the elastic walls of the air-cells. Hence chronic bronchitis, tuberculosis, general senile decay, and the like, operate as powerful predisposing causes of emphysema.

Given then, a sufficient decrease of the resisting power of the walls of the air-cells, the normal amount of tension constantly operating on them is quite sufficient to bring about emphysema in course of time—*senile* or *atrophous* emphysema for example. But obviously any condition which unduly increases this tension, at the same time renders the occurrence of emphysema still more certain. Such morbid increase of tension may in some cases occur during *inspiration*, and in others during *expiration*. For instance if air be prevented by disease from entering some part of the lung, it is clear that if the chest expand to the same extent as in health, the remainder of the lung will be over-distended (*vicarious emphysema*). Again, strong or sudden expiratory efforts with closure of the glottis, such as occur in coughing, straining, &c., must necessarily increase the pressure of the air within the lung at those parts of the chest cavity which are soft and yielding, such as at the apices, anterior borders, and intercostal spaces.

VARIETIES.—1. *Acute vesicular emphysema* is a common result of acute bronchitis and whooping-cough. It is due to the local malnutrition and violent cough met with in these diseases. As a rule it subsides with the condition from which it arose. The affected lobules are puffed up and pale; the capillaries are stretched and their network is enlarged.

2. *Chronic local emphysema* is characterised by extreme permanent over-distension of a few vesicles, and is most commonly associated with obsolescent tubercle of the apex of the lung. Its anatomical appearances resemble those of the acute form.

3. *Large-lung emphysema.*—Here the vesicles of the whole, or a large section of one or both lungs are over-distended, causing an increase both in the bulk of the lungs, and in the capacity of the thorax. The lungs are paler, larger, and drier than usual. They have a soft cushiony feel, and readily pit on pressure. If inflated they recoil to a less extent than usual. The vesicles are obviously enlarged, and not infrequently several are thrown into one by atrophy and perforation of intervening septa. This variety is generally associated with chronic bronchitis.

4. *Atrophous emphysema.*—This is essentially a disease of advanced life. Both lungs suffer equally, and are smaller, lighter, and drier than normal, and they shrink up on opening the chest. The walls of the air-cells are degenerated, atrophied, and often perforated.

Symptoms.—These are marked in direct proportion to the amount of lung-tissue affected. *Dyspnœa* is the chief direct symptom of emphysema, and is due partly to the destruction of the alveolar surface, partly to the difficulty of expiration, and partly to the rigid state of the chest walls. It is *expiratory* in character, owing to the impaired elasticity of the lung; and is aggravated by exertion, by flatulent distension of the abdomen, and by concurrent bronchitis. *Cough* is usually present, but is not attended with expectoration, except in those cases where there is co-existent bronchial catarrh. The complexion is dusky, and the patient is languid and dull.

Other symptoms of *indirect* origin are met with, which are mainly due to circulatory obstruction in the lungs from destruction, lengthening, and narrowing of the alveolar capillaries. This difficulty is at first counteracted by *hypertrophy* of the right ventricle; but sooner or later, symptoms of cardiac *dilatation* supervene, which will be described in another chapter.

The *physical signs* in a marked case of emphysema are as follows:—1. In the *hypertrophous* form, the chest is enlarged throughout, the ribs are raised, the shoulders high, the sternum thrust forward, and the dorsal spine curved outwards—the chest is "*barrel-shaped.*" In the *atrophous* form the chest is small and long, and the ribs are very oblique. 2. Expansion of the chest is deficient while elevation movements are in excess. 3. There is hyper-resonance on percussion. 4. The breath sounds are harsh or exaggerated and divided, and *expiration is prolonged.* 5. Râles (due to co-existing catarrh) are often present.

The *prognosis* as to complete recovery is unfavourable in chronic cases.

TREATMENT.—1. Attention to the cause of the disease and removal when possible. 2. Due regard for the proper action of the alimentary canal. 3. Medicinally, tonics and diffusible stimulants are indicated. Among the best are cod-liver oil, tincture of steel, arsenic, and quinine; which may be combined with ether, tincture of lobelia, or spirits of chloroform, if thought desirable. Morphia and other narcotics require caution in their employment. 4. Complications must be treated as they arise. 5. Change to a mild and equable climate is often beneficial.

PULMONARY COLLAPSE.

The term "*collapse of lung*," strictly speaking, refers only to lung-tissue which is deprived of air. Collapse is said to be *congenital*, when the lungs have been only imperfectly expanded at the time of birth (*atelectasis*); but it is more often *acquired* in after-life. The latter alone calls for description here. It may arise from *direct pressure on the lung*, as in copious pleural effusions; or more commonly as a result of *capillary-bronchitis* or *whooping-cough*—a very frequent occurrence with young children.

The mechanism of its production in the latter case, according to Gairdner, is as follows:—The bronchi contain plugs of mucus, which, partly owing to the gradual centrifugal diminution in the calibre of the air-tubes, and partly to the greater force of expiration over inspiration, admit the expulsion of air during expiration, but prevent its return during inspiration. But experiments show that wherever a bronchus becomes plugged from any cause, the air imprisoned behind the obstruction is quickly absorbed, and collapse follows; and the generalisation is probably true, that given persistent interference with the inflation of an area of lung-tissue from any cause, absorption of air and collapse invariably occur. Feeble inspiratory power and the small size of the bronchi sufficiently explain the greater frequency of collapse in children.

Collapse may be limited to a single lobule, or involve the entire lung. Collapsed lung-tissue is tough and leathery, sinks in water, does not crepitate on pressure, is of a dark slate colour, is depressed on its outer surface below the level of surrounding lung, and when of recent origin may readily be re-inflated. Collapsed lobules are, however, especially apt to undergo inflammatory changes; and in that condition can no longer be re-inflated.

The *symptoms* of collapse, when well marked, are characterised by considerable restlessness and prostration, a low temperature, dusky skin, dyspnœa chiefly affecting inspiration, persistent ineffective cough, and a feeble whining voice. With regard to *physical signs*, the lower ribs and epigastrium may be observed to sink inwards during inspiration; and there may be some dulness on percussion, with weak or bronchial breathing over the affected area. Slight degrees of collapse do not admit of diagnosis.

The *treatment* is that for the condition on which the collapse depends.

GANGRENE OF THE LUNG.

Gangrene is rare, but it sometimes supervenes on acute pneumonia and other diseases of lung; sometimes as the result of compression of the root of a lung by an aneurism or new growth; sometimes from the presence of decomposing particles of food and secretions in the air tubes, especially among the insane and intemperate; and sometimes as a sequel to pyæmic types of specific fevers, &c.

Anatomical Characters.—It occurs either as *circumscribed nodules* of variable size, or is *diffused* through a large part of the lung without any line of demarcation. The gangrenous tissue becomes softened, pulpy, of a greenish colour, and horribly fœtid.

Symptoms.—The peculiar and extremely *fœtid odour of the breath*, and the *expectoration of dirty-brownish or blackish sputa, often containing fragments of lung-tissue*, are very characteristic. Hæmoptysis sometimes occurs. There is great general depression, with low nervous symptoms, and a marked tendency to death from collapse. It is almost always fatal.

Physical Signs.—At first the percussion-note may be tympanitic, but it is afterwards dull. The breath-sounds are weak, and attended with moist râles. Sometimes we meet with the signs of a cavity.

It should be remembered that a gangrenous odour of the sputa is sometimes noted in certain cases of chronic empyema, or chronic bronchitis.

Treatment.—Abundant nourishment, and stimulants (alcoholic and otherwise) are always necessary. Antiseptic inhalations of creosote, carbolic acid, or turpentine are recommended. Quinine, with mineral acids, may be given internally.

CANCER OF THE LUNG.

Cancer of the lung is almost invariably secondary to cancerous disease elsewhere, notably in the stomach and breast. It is useful to recognise at least three forms in which it occurs:—(*a*) small disseminated nodules—generally of embolic origin; (*b*) large masses in one or both lungs—generally growing in from the mediastinal glands; and (*c*) nodules on the pleural surface of the lung—generally accompanied with serous or hæmorrhagic effusion.

Corresponding to the irregular distribution of the disease, there is great variety in the symptoms and signs to which it gives rise. In the disseminated form there are often no symptoms at all. At other times, we may meet with cough, dyspnœa, fever, and occasional hæmoptysis, without obvious dulness on percussion, closely resembling acute miliary tuberculosis of lung. Cancer, in mass, may sometimes be attended with little more than the ordinary signs and symptoms of mediastinal tumour. When, however, a large part of the lung is involved, it may yield physical signs which are almost indistinguishable from those of a pleural effusion. When cancer of the lung is soft and is growing rapidly, it is very apt to break down, and lead to the formation of a cavity. Such cases are attended with hæmoptysis, cough, dyspnœa on exertion, emaciation, fever, and the physical signs of localised pneumonia, and closely simulate phthisis. In any case the strength gradually fails, and the patient finally dies from exhaustion.

The treatment is purely palliative and symptomatic.

SUB-SECTION IV.

DISEASES OF THE PLEURA.

PLEURISY.

PLEURISY may be *acute* or *chronic*. Its most common cause is exposure to cold; but sometimes it arises from injury, or secondarily to adjacent inflammation, or in the course of some other disease, *e.g.*, Bright's disease or scarlet fever.

MORBID ANATOMY.—The membrane is first congested, and soon loses its usual polish by shedding its endothelium. Next a layer of coagulable lymph enclosing innumerable cells is thrown out on the surface, and a variable quantity of sero-fibrinous fluid accumulates within the pleural cavity. The elastic lung retracts as the fluid effusion increases, and *no direct pressure can be exerted by the fluid until the elastic retraction of the lung is* *exhausted.* In extreme effusions, however, the lung may be *pressed* backwards and inwards against the spine, becoming more or less "collapsed," or altered by inflammation. The opposite lung is collaterally congested. Similarly the heart is often displaced—at first by the elastic traction of the healthy lung on the mediastinum, and *afterwards* (in some cases) by direct pressure of the fluid. The diaphragm, liver and spleen are never displaced until the elastic retraction of the affected lung is exhausted.

In favourable cases the fluid is absorbed after a while, and the pleural surfaces come into contact with each other once more; the exudation of lymph disappears more or less completely, and what remains becomes organised into fibrous tissue, causing adhesions and agglutinations. Where, however, the lung is prevented from proper re-expansion by the resistance of the exudation, and the absorption of the effusion continues, the affected side of the chest sinks in, and the mediastinum with its contents is drawn over to the same side. But there is an obvious limit to this; and when it is reached without contact of the lung and the chest-wall being re-established, a chronic effusion remains.

The term *empyema* is applied to those cases in which the effusion is purulent, or afterwards becomes so.

SYMPTOMS.—The onset of acute pleurisy may be either insidious, or more or less sudden and marked by repeated chills. The temperature at once begins to rise, but rarely exceeds 102°, and not infrequently falls considerably short of this. Simultaneously, or shortly afterwards, pain, dyspnœa, and cough are observed. The pain is generally referred to the inferior mammary region on the affected side, and is sharp, lancinating, and especially aggravated by deep inspiration, or coughing. The dyspnœa is slight as a rule, and is mainly due to the pain in the side and the fever. In fact, when these have subsided, it is remarkable how little the breathing is disturbed, even when the chest is full of fluid, so long as the patient remains quiet. Marked or persistent dyspnœa generally indicates some pneumonic complication. The cough is short, hacking and accompanied with little or no expectoration. The decubitus varies much, but more commonly the patient lies on the affected side.

The temperature exhibits no definite course; the pulse is frequent, but its ratio to the respiration is only slightly disturbed; the tongue is somewhat furred; the appetite is impaired; thirst is increased; and the bowels are confined.

After a variable duration the attack terminates by recovery, with or without retraction of the chest-walls, according to the extent to which the lung is capable of re-expansion; by death (very rarely); or by passing into a chronic form.

Chronic Pleurisy.—Here the effusion persists, and may either remain sero-fibrinous, or become purulent (*empyema*). Purulent transformation sometimes takes place very early, especially in young children, or in those whose health is impaired. When the fluid is purulent there is always more or less febrile disturbance, usually of the hectic type. The skin of the affected side may be reddened, tender, swollen, or œdematous; the patient becomes notably anæmic; the finger ends are often clubbed; and night-sweats are not infrequent. The cough is troublesome, and may be accompanied with muco-purulent expectoration, which in rare cases possesses a gangrenous odour.

Complete recovery may take place by absorption of the fluid, by spontaneous discharge into the bronchi, or externally, or after the operation of paracentesis. In other cases the recovery is incomplete, owing to the persistence of lung mischief, or the establishment of a pleural fistula. Lastly, death may occur from asthenia.

Physical Signs.—*Before effusion*:—(1) Diminished movement of the affected side; and (2) friction-sounds. *During effusion*:—(1) Enlargement of the side and widening of the intercostal spaces; (2) absent or deficient vocal fremitus; (3) absent or deficient expansion of the side; (4) dulness and increased resistence on percussion over the area of the effusion, beginning at the base and extending upwards, together with a tympanitic percussion-note under the clavicle of the same side; (5) weak or absent breath-sounds, which sometimes exhibit a bronchial quality, especially in children; (6) absence of friction-sounds previously heard; (7) weak or absent vocal resonance over the fluid, with ægophony (in some cases) best heard about the upper margin of the effusion; and (8) signs of cardiac displacement. *During absorption* the above signs gradually disappear, and the friction-sounds return for a while. Should the lung remain unexpanded, the chest-walls retract, the ribs become very oblique, the intercostal spaces very narrow, the shoulder on the same side falls, and the angle of the scapula projects. Impaired movements, some dulness, and weakness of the breath-sounds on the affected side, usually persist for a considerable time.

Primary pleurisy is far more favourable than secondary pleurisy. The chronic form is apt to end unfavourably, though not often in itself the immediate cause of death. Serious collateral congestion of the opposite lung constitutes a frequent source of danger.

The *treatment* at the onset should consist of rest in bed, hot and moist applications to the chest, with small doses of aconite—frequently repeated—given internally. Dr. Roberts strongly recommends firm strapping of the affected side from the onset, with a view of rendering it immobile. The proper action of the skin, bowels, and kidneys, should be promoted. To relieve the pain—subcutaneous injections of morphia, or a few leeches may be employed with advantage in obstinate cases, To induce absorption of the effusion—counter-irritation by means of flying-blisters, stupes, sinapisms, or croton oil liniment, together with the administration of iodide of potassium, or pills containing mercury squills and digitalis in combination, have been recommended; but the action of any mercurial should be closely watched. Paracentesis thoracis, or tapping the chest, may be resorted to for the removal of the fluid; and the operation is indicated where the dyspnœa is urgent, when syncope threatens, when the fluid is purulent, or when the effusion has become chronic. The methods for its performance include simple puncture with trocar and canula, aspiration, and free incision. The wound after a first operation should, as a rule, be closed and sealed up; but when the fluid is purulent or fœtid, it is generally desirable to keep up drainage for some time afterwards. When the trocar and canula are employed, it is prudent to maintain a spray of carbolic acid before the opening while the fluid is being discharged. In chronic cases of empyema with great retraction of the side, and obliteration of the intercostal spaces, excision of a portion of a rib has been practised with success in children.

The diet should be liberal and nutritious; but stimulants are not usually necessary except in chronic cases where the strength is failing, when alcohol and tonic remedies are always indicated.

HYDROTHORAX—DROPSY OF THE PLEURA.

Hydrothorax only occurs as a part of general dropsy, and hence is generally associated either with Bright's disease, or heart disease. It for the most part presents the symptoms and signs of pleurisy with effusion ; but it differs from the latter by the absence of all fever, its occurrence on *both* sides of the chest, and the lower specific gravity of the fluid.

The only *symptom* to which it gives rise is dyspnœa, which is marked in proportion to the amount of effusion. The *physical signs* are those of the effusive stage of pleurisy ; but there is no friction-sound at any time. Its *treatment* is that of the affection on which it depends ; but when urgent symptoms are traceable to the effusion, paracentesis may be performed with advantage—preferably by aspiration.

HÆMOTHORAX—HÆMORRHAGE INTO THE PLEURA.

Hæmorrhage into the pleural cavity in any quantity is usually the result of bursting of an aneurism, or the rupture of a vessel from injury. It is well to recollect that hæmorrhagic serous effusion, with all the signs of ordinary pleuritic effusion, almost invariably indicates the presence of some new growth in the pleura, such as tubercle or cancer.

PNEUMOTHORAX—AIR IN THE PLEURA.

Air in the pleura is almost always the result of the rupture of a phthisical cavity of the lung. Sometimes however, it arises from a wound of the chest-wall. The entry of air is at once followed by retraction of the lung on the affected side, as far as any diseased condition which may happen to be present will admit; and for similar reasons traction of the opposite lung on the mediastinum may cause displacement of the heart. Pleurisy soon follows, with the effusion of a serous or more often a purulent fluid. When the opening into the pleural cavity is free, the pressure of the contained air corresponds with that of the atmosphere; but when, as the result of a valvular opening, air is drawn in during inspiration and cannot be expelled during expiration, the pressure of the contained air may be much raised.

SYMPTOMS.—The symptoms commence suddenly—often during an attack of coughing—with pain in the side, a sense of something having given way, urgent dyspnœa, and general collapse. The pulse becomes more frequent, and is weak and small; the temperature is apt to fall at first; and the patient has an anxious appearance, and is often unable to lie down.

The *physical signs*, for reasons already given, are those of both fluid and air in the pleural cavity. The chest is enlarged on the affected side, and its movements are diminished; the vocal fremitus and resonance are weak or absent; the percussion-note is tympanitic at first, but as fluid collects, this gives place to dulness at the most dependent part of the chest, the limits of which alter with change of posture; the breath-sounds are weak or absent, but when the wound is open it is often typically amphoric; metallic tinkling is often observed; and not infrequently a splashing sound may be obtained on sharp succussion or shaking of the patient.

The *treatment* in the main is that of pleurisy. A subcutaneous injection of morphia is often of great service at the outset, to diminish the effects of the shock and to relieve the pain. Later on, half-drachm doses of ether given every hour are useful. Alcohol often increases the pain. Locally, nothing is better than firmly strapping the affected side.

Phthisical pneumothorax is generally fatal, and sometimes very rapidly so. More often the patient lingers for a week or two. Traumatic cases without serious complicating disease may recover.

Section V.

DISEASES OF THE CIRCULATORY SYSTEM.

Sub-Section I.

DISEASES OF THE HEART AND PERICARDIUM.

TABULAR STATEMENT OF PHYSICAL SIGNS.

I. Inspection.

Pulsation . . .	Cardiac impulse . .	Position. Extent.
	Abnormal pulsation	Position. Extent.

II. Palpation.

1. Pulsation . .	Cardiac impulse . .	Position. Extent. Quality.
	Abnormal pulsation .	Position. Extent. Quality.
2. Valvular thrills	Position of maximum intensity.	
	Rhythm	Systolic. Diastolic. Pre-systolic.
3. Friction-fremitus.		
4. Fluctuation (?).		

III. PERCUSSION.

Area . . .	Cardiac region . . .	Area increased.
	Mediastinum . . .	Area diminished

IV. AUSCULTATION.

Cardiac	Sounds	Intensity. Rhythm. Quality.
	Murmurs	Point of maximum intensity. Rhythm. Direction of conduction.
Pericardial . . .	Friction-sounds.	

V. THE PULSE.

Observe its . .	Frequency	Increased. Diminished.
	Rhythm	Regular. Irregular.
	Force	Equal. Unequal.
	Volume	Large. Small.
	Tension	High. Low.

DISEASES OF HEART-SUBSTANCE.

HYPERTROPHY.

HYPERTROPHY of the heart denotes an increase in the thickness of its walls, as a result of an increase in its muscular elements. Two varieties are recognised, according as the cavities are dilated or of normal size. The former is termed *eccentric* hypertrophy; the latter *simple* hypertrophy. Pure hypertrophy cannot be rightly regarded as a disease *per se;* it represents a compensatory adaptation on the part of the heart, rendered necessary by *increased work.* The circumstances which lead to increase of work fall into one of two groups:—(1) An increase in the volume of blood to be propelled, arising from dilatation of the heart-cavities; or (2) an increase in the peripheral resistance to be overcome, as the result, for instance, of stenosis of a valvular orifice, oft-repeated laborious exertion, emphysema, or vascular thickening and narrowing such as occurs in some cases of chronic nephritis. In previous editions of this work, long-continued nervous excitement of the heart, such as happens in Graves' disease or from abuse of tea and tobacco, was mentioned as a possible cause of hypertrophy; but further observations have led me to conclude that in these cases hypertrophy is preceded by cardiac dilatation, and is dependent upon it. The heart is increased in weight and bulk. Its shape also is modified according to the distribution of the hypertrophy. The latter necessarily varies in its seat and extent according to its cause, but the left ventricle is most frequently affected.

The special effect of hypertrophy upon the circulation is that the arteries become fuller, the veins less full, and the circulation is accelerated. Thus, hypertrophy of the right heart tends to the overfilling of the *pulmonary* arteries, and the emptying of the *systemic* veins; while the reverse obtains in hypertrophy of the left heart. It will be understood, that whereas hypertrophy represents a process of compensation, these results are neutralised in the defects to be made good. When it is otherwise, we may expect to find turbulent action of the heart, dyspnœa, a sense of oppression in the chest, headache, flushing of the face, flashes in the eyes, noises in the ear and giddiness.

From this point of view it is difficult to suppose that hypertrophy gives rise to morbid symptoms; and if any such symptoms are present, they probably result either from imperfect hypertrophy, or incipient dilatation.

The physical signs of hypertrophy of the left ventricle are precordial bulging (occasionally), diffusion and displacement of the apex-beat downwards and outwards, slow heaving impulse, enlargement of the area of cardiac dulness, prolongation of the first sound, accentuation of the second sound, and a full and tense pulse. The signs of hypertrophy of the right ventricle are not so marked. The apex-beat is displaced to the left but not downwards; well marked pulsation at the epigastrium, and sometimes in the third and fourth rib-interspaces; and the cardiac dulness is increased to the right.

The powerful action of the heart in hypertrophy necessarily involves unusual mechanical disturbance and irritation of the valve segments and arterial walls (especially in the aorta). This in turn favours atheroma and the occurrence of aneurism.

DILATATION.

Dilatation obviously implies the enlargement of the heart-cavities, the walls of which may be either thinner than normal or hypertrophied. The proximate physical cause of dilatation is invariably the same—namely distensile strain brought to bear upon the walls of the heart when in a state of relaxation during diastole; and this strain depends in its turn on an overloading of the affected chamber with blood. This may be brought about in one of two ways. In the first place, there may be direct overloading, such as occurs for instance in the case of the left ventricle, when the aortic valves are incompetent. During diastole the ventricle obviously becomes overloaded in direct proportion to the amount of aortic regurgitation, and in the same degree becomes over-distended. Accordingly dilatation is an invariable feature of aortic incompetence. A second and much more frequent cause of over-loading and over-distension consists in the inability of the heart to completely empty its cavities, either from inherent weakness on the part of the heart-wall or from an amount of peripheral obstruction to be overcome in excess of its power. The heart-cavities clearly must subsequently become overloaded and over-distended with blood in direct proportion to the quantity which remains behind. And since the resistant power of the walls of a weak heart is very commonly also reduced, we see in this fact another condition favourable to distension from slight causes. It is to this weakness, incomplete emptying, and dilatation of the heart, that the symptoms of all cardiac diseases are mainly attributable, in the first instance; and, accordingly dilatation acquires a pre-eminent importance in cardiac pathology.

Symptoms.—The general effect on the circulation is that the veins become engorged, the arteries imperfectly filled, and the circulation as a whole is retarded. The earliest symptom of dilatation is palpitation, representing a tumultuous struggle on the part of the heart with work to which it is for the time unequal. Dyspnœa follows closely after from interference with the pulmonary circulation; while still later, we meet with cyanosis, dropsy, and a variety of other symptoms directly or indirectly due to mechanical congestion of the parts concerned.

The chief physical signs are—diffusion of the apex-beat which has a jerking or slapping quality, and is often irregular or undulating; increased area of cardiac dulness, especially towards the right side; short sharp clear heart-sounds, and a feeble small and often irregular pulse. When hypertrophy is combined with dilatation, as so often happens, the signs of each are correspondingly mixed.

The treatment of dilatation is practically identical with that required for chronic valvular disease, and its consideration will be deferred to that chapter.

MYOCARDITIS.

The muscular substance is occasionally the seat of acute or chronic inflammatory change. It is usually secondary to inflammation of the endo- or pericardium, but it sometimes occurs in pyæmia, or typhus fever. In acute cases, either the muscular substance generally becomes softened and degenerated, or it presents foci of suppuration here and there. Chronic inflammation, on the other hand, leads to fibroid infiltration and induration. The symptoms are those of cardiac failure, *e.g.*, palpitation, dyspnœa, œdema, cyanosis, irregular and intermittent pulse, and the like; but the diagnosis is always most uncertain.

FATTY DEGENERATION OF THE HEART.

Fatty degeneration of the heart is the result of chronic malnutrition of the heart's substance. It sometimes forms a part of more or less general failure of nutrition from senile decay, long-standing anæmia, or chronic disease; sometimes it is traceable to interference with the coronary circulation, arterial degeneration, cardiac dilatation, endo- or pericarditis; and it may also be induced by phosphorus and certain other kinds of poisoning. Chronic alcoholism, gout, and sedentary habits are also common predisposing causes. It generally occurs in the latter half of life.

The *anatomical changes* are unequally distributed through the organ, and the whole thickness of the cardiac walls is not necessarily affected. The left ventricle is its most common seat. The muscular substance is paler than normal, breaks down readily on pressure, loses its fibrous appearance, and has a greasy feel. The individual fibres gradually lose their striæ, become converted into fat-granules, and finally disappear altogether. The process now described must be distinguished from infiltration and encrustation of the heart with fatty tissue, though the two are sometimes associated.

SYMPTOMS.—The diagnosis of fatty heart is never very easy, it is generally very difficult, and sometimes impossible. In a typical case, however, the countenance is pale and anxious, the temper becomes irritable, the extremities are chilly (especially at night), perspirations break out from slight causes, and sleep is disturbed. The breathing is oppressed and shallow, especially on exertion; and involuntary sighing is often observed. The heart's impulse is weak; and the pulse is feeble, slow (as a rule), and is often persistently irregular both in force and rhythm. From time to time, the patient is subject to sudden attacks of faintness, giddiness, or dyspnœa, with or without pain at the chest, in any of which he may die outright. A large proportion of sudden deaths arise from this cause. The condition is always serious, when well-marked. It sometimes leads to rupture of the heart.

TREATMENT.—The subjects of fatty heart should lead a quiet, even, and uneventful life, sudden efforts at any time being especially dangerous. At the same time gentle exercise in the fresh air may be taken, provided that it always stops short of fatigue. The diet should be light, nutritious, and mildly stimulating; and a small quantity of light wine daily, is, as a rule, desirable. Medicinally, arsenic and preparations of ammonia and iron, in combination with digitalis or nux vomica are of most value. The bowels should never be permitted to become stubborn and confined.

ACUTE ENDOCARDITIS.

THIS affection rarely occurs except as secondary to other diseases, especially rheumatic fever and Bright's disease. It is most common among the young.

MORBID ANATOMY.—The endocardium first becomes congested and loses its natural polish by shedding its epithelium. This is followed by the formation of little prominences of granulation-tissue (*vegetations*) which occur for the most part on the contact-surfaces of some one or more valve-segments, or on the *chordæ tendineæ.* Such vegetations generally increase in size and sometimes form large fungating excrescences, which project into the heart cavity, and may obstruct or otherwise interfere with the circulation. These changes are most commonly met with on the left side of the heart, especially affecting the mitral orifice and valves. The most important ultimate results of endocarditis are (1) thickening and puckering of the valve-segments; (2) adhesion of the valve-segments to each other; (3) thickening, shortening and agglutination of the *chordæ tendineæ;* and (4) narrowing of the orifices.

The chief *symptoms* are palpitation, increased frequency of the pulse, dyspnœa, and pyrexia. The symptoms, however, are always vague and indefinite, until the orifices or their valves become involved, which is generally the case.

The *physical signs* indicate cardiac excitement and valvular disease, including increased force and area of apex beat, and valvular murmurs. Disease of the mitral orifice and valve is most common, that of the aortic orifice and valve coming next in frequency. The right heart is comparatively rarely affected.

The *treatment* chiefly resolves itself into the treatment of the diseases with which the endocarditis is associated, and the adoption of certain principles which will be alluded to in the next chapter. Counter-irritation, or warm and moist applications to the precordia, with the administration of cardiac sedatives internally, have been recommended.

ULCERATIVE ENDOCARDITIS.—This term is applied to certain cases of endocarditis, which are characterised by destructive endocardial changes, and peculiar symptoms of a septic type. The margins or faces of the valves become eroded at one or more points, forming rough irregular ulcers, which may be followed by perforation or aneurism of the valve-segment, or rupture of the chordæ tendineæ. Micro-organisms are found in the ulcerating tissue. Infarcts are of frequent occurrence in various organs; and being of an infective nature, are followed by suppuration (*metastatic abscesses*).

The symptoms are those of pyæmia, and are due to the passage of septic matter from the endocardial lesion into the blood. The course is in some cases very acute, while in others it is comparatively chronic, closely resembling a lingering form of typhoid or remittent fever. The physical signs are those of ordinary endocarditis. The termination is almost always fatal. Little can be hoped from treatment; but large doses of quinine, with a liberal supply of nutritious food and stimulants would appear to be most desirable.

CHRONIC VALVULAR DISEASES OF THE HEART.

VALVULAR disease may arise from (1) endocarditis; (2) atheromatous and calcareous degenerations; (3) enlargement of the orifices, due to excessive cardiac dilatation; or (4) congenital malformation. Disease of one orifice or set of valves is very apt to extend to others.

The proximate effect of valvular disease is either to cause (1) *insufficiency*, which, owing to a disproportion between the size of the orifice to be closed and the valves to close it, gives rise to *regurgitation* of the blood current; or (2) *obstruction*, which, owing to constriction or partial blocking of the orifice, impedes the onward flow of blood. Each orifice is theoretically liable to either kind of disturbance, *regurgitant or obstructive*.

The general progress of chronic valvular disease exhibits a broad uniformity, whatever may be the nature of the initial valvular lesion. Four stages may be recognised. In the *first stage* the troubles are chiefly of a *physical* kind. The heart is unable to empty its cavities completely, and becomes (together with the large veins which enter it) over full, while the arteries which issue from it are imperfectly filled. Under favourable conditions (especially in disease of the left heart) these defects may be overcome more or less, by compensatory hypertrophy of those chambers of the heart on which the strain falls; and with complete compensation all symptoms disappear. Compensation, however, is rarely complete, is often very imperfect, and sometimes does not occur at all. In no case is compensation permanent, supposing the patient does not die from intercurrent disease. After a variable time, it breaks down, and symptoms of cardiac failure begin with palpitation and dyspnœa; something—that is to say—occurs, as a result of which the heart becomes dilated, and is no longer able to empty itself completely. The causes of such failure vary, but among the commonest are over-work, malnutrition, nervous exhaustion, intercurrent disease, abuse of alcohol, increase or extension of the valvular lesion, too rapid growth, pregnancy, over-lactation and senile decay.

The *second stage* of chronic valvular disease, in addition to the physical troubles already mentioned, is characterised by *a series of grave functional disorders resulting from mechanical congestion* of the parts concerned—disorders of digestion, of absorption, of assimilation, of blood-making, of secretion, and of elimination. The liver and the dependent lower limbs are the first to suffer, because of the low blood-pressure in these parts. When the primary lesion is localised in the right heart, or when compensation is impossible, either by reason of feeble vitality, or the magnitude of the defect to be overcome, the case enters upon the second stage from the beginning.

Sooner or later a *third stage* supervenes, which is characterised by the super-addition to previous troubles of *permanent structural changes* in various organs from prolonged congestion, notably in the liver and kidneys. The *fourth and last stage* is manifested by *general cachexia* from long-standing interference with important vital functions; and, anatomically, by degeneration—notably in the muscular tissue generally, and that of the heart in particular.

Mitral Regurgitation.—This is one of the most common valvular affections, and inasmuch as it commonly arises in connection with rheumatic fever, it is met with most frequently in early adult life. It may result from endocarditis, from simple valvulitis supervening on aortic disease, or from dilatation of the left ventricle.

Morbid Anatomy.—The chief alterations met with are thickening, puckering, and rigidity of the valve-segments; atheroma and calcification; adhesion of the valve-segments to each other; or shortening, thickening or rigidity of the chordæ tendineæ. The right auricle and ventricle are more or less hypertrophied, with or without dilatation.

Clinical History.—At each left ventricular systole, blood regurgitates into the left auricle, the pulmonary circulation thus becomes over-full and is subjected to increased pressure. This difficulty is met for a time by hypertrophy of the left auricle and right ventricle. Hence, during this stage there are few if any manifestations of discomfort; but at the same time it should be borne in mind, that owing to the increased tension of blood in the lungs, there is not infrequently some dyspnœa on exertion, and any slight intercurrent attack of catarrh is apt to become serious. Sooner or later compensatory hypertrophy gives place to dilatation, and the engorgement and increased tension of the pulmonary vessels are reflected backwards *through* the right heart to the systemic veins, which now in turn become overloaded; the aorta is underfilled, and the circulation is retarded. The surface of the skin is dusky or livid; the ankles become œdematous and the anasarca gradually extends upwards; cough and dyspnœa are well marked, especially on exertion; and the expectoration is profuse, watery, and sometimes contains blood. The further progress of the case conforms to the general description already given. The *physical signs* are (1) a *murmur* of maximum intensity at the left apex, occurring with or replacing the first sound, and conducted outwards into the left axilla; (2) accentuation of the second sound at the base of the heart to the left of the sternum; (3) an occasional systolic thrill over the apex; (4) a small, unequal pulse, often out of all proportion to the apparent force of the heart beat.

Mitral Obstruction is nearly always due to endocarditis and its effects.

Morbid Anatomy.—The orifice is constricted or narrowed, and its margins are often irregular and thickened. The valve-segments frequently adhere together, producing a funnel-shaped passage into the left ventricle, which terminates in a narrow slit-like opening *(button-hole mitral disease)*. The left auricle and right ventricle are hypertrophied or dilated.

The *clinical history* is similar to that of mitral regurgitation, with which it is usually combined.

The *physical signs* are (1) *a murmur* of maximum intensity at the left apex, running up to and ending with the first sound (*pre-systolic*) and not markedly conducted in any special direction; (2) a pre-systolic thrill which is almost always present; (3) accentuation of the second sound at the base; and (4) a small and feeble pulse.

Aortic Regurgitation constitutes one of the most serious valvular lesions; and may be caused by chronic inflammation with degeneration of the valves, rupture of a valve-segment, or more rarely by endocarditis. It occurs most frequently in the latter half of life.

Morbid Anatomy.—The valve-segments are puckered, thickened, or adherent, and occasionally they are ruptured, perforated, or lacerated. The left heart is greatly enlarged by hypertrophy and dilatation.

Clinical History.—The general effect of aortic regurgitation is to retard the circulation. The increased strain brought to bear upon the left ventricle during its diastole causes dilatation from the first, but in order to meet the demand for extra propulsive work caused by the increased contents of the cavity, hypertrophy soon develops; and, if the amount of regurgitation be slight, and the general nutrition of the heart-wall be good, approximate compensation may take place. More often, however, dilatation slowly progresses, and hypertrophy stops short of complete compensation. Thus, palpitation, dyspnœa on exertion, headache, and vertigo, occur in the early stages. Sooner or later, the mitral valve becomes incompetent, and a train of symptoms then sets in, similar to what has already been described under the head of mitral regurgitation. If the inflammatory or atheromatous changes extend from the aortic orifice to the coronary vessels, the nutrition of the heart-muscle becomes rapidly impaired, and degeneration takes place, thus accelerating the progress of the case towards an unfavourable issue. Death generally occurs from cardiac failure, syncope, apoplexy, or embolism.

The *physical signs* are (1) a more or less heaving and diffused impulse, the apex beat being always displaced downwards and outwards; (2) increased length and breadth of the cardiac dulness; (3) *a murmur* of maximum intensity at the base, occurring with or replacing the second sound, conducted downwards towards the xiphoid cartilage or apex, and also often heard in the back; (4) an occasional diastolic thrill at the base; (5) *a sudden jerking pulse* with unusual pulsation of the carotids. This lesion is very frequently combined with aortic obstruction. The arteries generally are apt to become tortuous, elongated, and resistant, from degenerative changes.

Aortic Obstruction.—The etiology of this lesion is similar to that of aortic regurgitation.

Morbid Anatomy.—The valves may become rigid, thickened and adherent, so that they cannot be pressed back; or the orifice as a whole may be puckered and narrowed. In no form of cardiac valvular disease is pure hypertrophy of the left ventricle better marked.

Clinical History.—In the early stages the symptoms are usually *nil*, for the compensatory hypertrophy of the left ventricle is very complete and persistent, and advantageously occurs *immediately in the rear* of the lesion for which compensation is needed. After awhile the mitral orifice with its valves may become implicated, and then the former equilibrium breaks down. Or again, owing to the frequency with which it is associated with aortic regurgitation, symptoms of discomfort may be present from the outset. The symptoms therefore respectively correspond with those of mitral and aortic regurgitation already described.

The *physical signs* are (1) a heaving diffused impulse which is displaced downwards and outwards; (2) increased length and breadth of cardiac dulness; (3) a murmur of maximum intensity at the base, occurring with or replacing the first sound, and conducted upwards towards the large arteries of the neck; (4) an occasional systolic thrill at the base; and (5) a small, steady, but usually regular pulse.

TRICUSPID REGURGITATION.—This occurs either as the result of dilatation of the right heart, or of vulvulitis. It is generally the sequel of valvular disease on the left side of the heart; but it not infrequently results from long-standing obstruction in the pulmonary circulation from bronchitis and emphysema. Its morbid anatomy does not differ essentially from that met with in lesions of the left auriculo-ventricular orifice.

Clinical History.—At each ventricular systole the blood is thrown through the right auricle into the venæ cavæ and jugulars, which speedily become dilated and overloaded. A *reflux pulsation of the jugulars*, which is synchronous with each ventricular contraction, is pathognomonic of tricuspid regurgitation. It therefore follows that the symptoms which arise from mechanical systemic congestion, come on quickly and in an exaggerated form. (See *Mitral regurgitation*).

The chief *physical sign* of importance is a *systolic murmur*, which is of maximum intensity over the ensiform cartilage. It is not invariably present, and when associated with a mitral systolic murmur, as so frequently happens, their differentiation is very difficult and often impossible.

Tricuspid obstruction and *pulmono-arterial valvular disease* are of such extreme rarity that no special description of them is required in a work of this kind.

Diagnosis of Valvular Disease.—The diagnosis of valvular disease is mainly based upon the existence of a *murmur.* An exocardial murmur or friction-sound is sometimes liable to be mistaken for an endocardial murmur. Friction-sound, however, is mainly distinguished by its "to and fro" rhythm, its superficial grazing or rubbing character, distinct localisation, and absence of any obvious relation to the cardiac sounds. Again, all endocardial murmurs are not due to organic disease of orifices and valves. Murmurs of functional origin occur in anæmia as already described; and dilatation of the left or right ventricle may give rise to mitral or tricuspid regurgitant murmurs respectively, without any structural change in the endocardium. Classification of cases can only be made (1) by a very careful physical examination; (2) by a full enquiry into the previous history, with especial reference to rheumatic fever and Bright's disease, not omitting sex, age, and nature of employment; (3) by a special examination of the superficial arteries and veins for irregularity, intermittence, abnormal pulsations, and indications of atheroma; and (4) by evidence or otherwise of circulatory disturbance, *e g.*, cyanosis, dropsy, &c.

Elements of Prognosis in Valvular Disease.—The gravity of a lesion bears no relation either to the intensity or to the quality of a murmur. Functional is less grave than organic disturbance. The nature of the conditions with which a valvular affection is associated or on which it depends, the habits and the employment of the patient, and the presence or absence of pulmonary renal or other complications, are of great importance. The condition of the walls of the heart and of the arteries demands the closest attention. Severe attacks of palpitation and dyspnœa on the least exertion, a feeble undulating impulse, an irregular or intermittent pulse, a liability to syncopal attacks, very scanty and albuminous urine, extensive anasarca serve to indicate weakness and degeneration of the former, while inspection and manipulation of the vessels, the age, and the presence or not of an *arcus senilis* give a clue to the condition of the latter. With reference to the relative seriousness of different varieties, it may be said that valvular disease of the right heart is more grave than that of the left; while on the left side, the gravity of aortic regurgitation, mitral regurgitation, mitral obstruction and aortic obstruction is in the order enumerated. Sudden death is not a common occurrence, but is most frequent in aortic regurgitation, and is generally due to syncope. Usually death takes place more gradually, as the result of secondary systemic disturbance.

TREATMENT OF VALVULAR DISEASE.—It should be our object to encourage hypertrophy in every possible way; and on this account, everything should be done which tends to improve the general vitality of the patient. When symptoms of cardiac failure supervene our primary duty is to enquire into and attend to the etiological indication. Thus we must rest a heart that has been overworked, feed a heart that has been starved, soothe a heart that has been worried, enjoin rigid teetotalism in those addicted to drink, interdict the use of tobacco in those who are depressed by resorting to it in excess, adjust the diet in those who have been pampered and over-fed, and so on. But of course while some of the proximate causes of cardiac failure are readily accessible and removable, others are only partially so, and the remainder are beyond control. At the same time, the demands upon the physical powers of the heart should be reduced to a minimum, by rest in the recumbent posture in proportion to the gravity of symptoms; always, however, bearing in mind that so soon as a circulatory equilibrium is realised, and symptoms subside, further rest will only weaken the system, and with it the heart also. It then becomes necessary to encourage the development of cardiac power by carefully graduated exercise. The chief precautions to be observed are these:—(1) not to attempt it until circulatory equilibrium has been established for the patient when at rest; (2) to be moderate at first; (3) to increase the amount gradually; and (4) always to stop short of fatigue. Moderate palpitation and oppression of breathing on beginning treatment need not be seriously regarded.

The food should be light, nutritious, digestible, and not too fluid. No heavy meal should be taken late at night. Alcohol is not needed as a rule in the early stages; but later on, it may be necessary for digestive purposes, for dyspnœic attacks, extreme feebleness of circulation, or restlessness at night. Tobacco smoking is often harmful, and tea and coffee should only be taken in moderation.

Medicinally, cardiac tonics, of which *digitalis* is the chief, are often most useful. It may be given in the form of tincture, infusion, or powder. In favourable cases, the pulse becomes slower, stronger, and more regular under its use, and the quantity of urine is increased. Sometimes it does not agree, but it is difficult to lay down precise rules for universal guidance. Iron, ammonia, strychnia, and vegetable tonics may be sometimes advantageously combined with digitalis. Belladonna, caffein, and convallaria, are also useful drugs, and are specially indicated when respiratory failure threatens. The bowels should be kept fairly open. The dropsy and turgid venous system which characterise the second stage of valvular disease suggest a further indication for treatment—*reduction of the fluids of the body*. If the condition is moderate and recent, a brisk hydragogue purge will often answer well. In other cases acupuncture of the swollen legs may be required. Periodical diaphoresis—preferably by subcutaneous injection of pilocarpine gr. $\frac{1}{12}$ to $\frac{1}{20}$—is also most useful, if carried out with caution and discrimination. It is desirable also to restrict the amount of fluid ingesta, unless the kidneys are much affected.

When once secondary structural disease has begun, and *a fortiori*, if there is much degeneration, treatment can only be palliative, and mainly consists of avoidance from undue exertion, gentle but systematic massage to keep up tissue nutrition, good feeding, and tonic remedies such as iron and arsenic.

The acute attacks of palpitation and dyspnœa (cardiac asthma) which not infrequently come on at night, are best combated by large hot poultices or fomentations to the chest, and free administration of sal-volatile, liquor ammoniæ, or spirits of chloroform in camphor water, with the addition of a few minims of digitalis or nux vomica. Hot brandy and water is also very useful in an emergency of this kind. When there is much cardiac excitement and pain, sedative drugs—such as bromide of potash, opium and the like—often answer better in the first instance than anything else, especially in cases of aortic disease. Opium, however, is contra-indicated where there is any serious interference with the processes of elimination.

NEUROSES OF THE HEART.

DISTURBANCES OF ACTION.

UNDER this head, palpitation, increased frequency, undue slowness, irregularity, and intermission will be considered. Each is, strictly speaking, a neurotic affection of the heart, under whatever circumstances it may occur; but there are good reasons in actual practice for recognising two varieties of each condition, according as the nervous disturbance is due to local cardiac disease (*organic*), or to causes operating at a distance from the heart (*neurotic*). Thus it is usual and useful to distinguish organic from neurotic palpitation, and so with regard to each of the other forms of disturbance mentioned. Speaking broadly, all organic disturbances of action point to more or less serious incapacity on the part of the heart to do its proper work; while neurotic disturbances, though of course morbid in nature, and sometimes indicative of grave trouble, are often compatible with average health and longevity.

Palpitation may be defined as the conscious sensation of tumultuous action of the heart. It varies from a mere feeling of fluttering, to agitation of the most distressing violence. It should be remembered that the amount of distress is no safe guide either as to the propulsive force of the heart nor to the state of the heart itself. It may be associated with choking sensations in the throat (globus), and a feeling of oppression at the chest. The frequency of the heart's action is increased. Palpitation is one of the earliest symptoms of cardiac dilatation in whatever way the latter may be brought about. It is also a common result of over-exertion, especially when associated with excitement. Under these circumstances it always indicates a demand of work from the heart in excess of its powers. In other cases it is due to central nervous disturbance from hysteria, emotional excitement, abuse of tea, coffee or tobacco, or irregular gout; or to reflex disturbance from flatulent dyspepsia, acidity of the stomach, uterine or ovarian disease, worms, or constipation. Persistent or frequently recurring palpitation usually points to dilatation.

Increased frequency of the heart's action may, like palpitation, be due purely to nervous disturbance, but under these circumstances, is only of temporary duration. Fever is a common cause of persistent increased frequency. In the absence of any rise of temperature, persistent increased frequency always points to cardiac weakness, either the result of local mischief or as part and parcel of general debility.

Undue slowness without any other sign or symptom of disturbance is of little importance, and in most cases cannot be adequately explained, and can only be regarded as an idiosyncrasy. Sometimes it is due to reflex stimulation of the cardio-inhibitory centre in the medulla, most frequently arising from irritation in the stomach or bowel, or again it may be due to central nervous disease. Lastly, we meet with undue slowness in connection with organic disease or fatty degeneration of the heart, but the circumstances of its occurrence are not properly understood.

Irregularity of rhythm also exhibits both organic and neurotic varieties. Of these conditions, the former is the more serious and the more common. It generally supervenes sooner or later in the course of all chronic valvular diseases, and marks the point at which the heart-substance has begun to degenerate. Neurotic irregularity is not uncommon in the gouty habit, or from abuse of tea or tobacco.

Analogous statements apply to *intermission.* Organic or "false" intermission (as it is sometimes called) is due to heart-beats so incomplete and ineffective that they are insufficient to cause a perceptible pulse-wave; and it indicates a more advanced degree of cardiac weakness than irregularity alone. "True" or neurotic intermission consists of complete occasional suppression of one or two beats, and occurs most commonly in connection with the gouty habit.

The chief characteristics of neurotic and functional cardiac disorder, as contrasted with that of organic origin, are marked disproportion between the subjective symptoms and the signs of disturbed circulation, the liability to assume a paroxysmal form, and the usual absence of any marked increase of symptoms on exertion. Moreover, while functional disturbance is more common in young adults of neurotic temperament, organic disturbance occurs more often after middle life, and has no relation to temperament.

The physical signs of structural cardiac disease are also usually wanting in cases of neurotic disturbance. With regard to this point two cautions are necessary:—(1) That cardiac degeneration may exist with little or no evidence on physical examination, and (2) that sometimes functional trouble may co-exist with organic disease, without any direct causal connection between them.

During a paroxysm of neurotic disturbance the patient should be removed from all sources of excitement. The local application of cold to the precordia, and the internal administration of bromide of potash, diffusible stimulants, or anti-spasmodics, or a hypodermic injection of morphia, or the inhalation of a little chloroform may be useful. The preventive treatment requires a careful study of the probable causes with a view to their removal when this is possible.

Cardiac Pain. Angina Pectoris.

Pain in the region of the heart occurs in all degrees of severity, from a momentary sharp twinge to a paroxysm of great agony. In its simplest form it is sometimes met with in feeble and anæmic subjects of a nervous temperament, and for the most part in early adult life. It consists of recurrent sharp stabbing pains in the precordia, and is occasionally accompanied with more or less persistent sub-sternal aching or soreness. Precisely similar symptoms are occasionally met with in those who take tea or coffee, or smoke tobacco in excess. Such attacks should be regarded as a simple neuralgia of the cardiac plexus, and are probably immediately excited by palpitation and irregular action of the heart. In some cases the irritation is reflected from the cardiac plexus through the spinal cord to one of the left intercostal nerves (usually the third or fourth), causing pain in the side.

Pain is not a common symptom of organic cardiac disease except in lesions at the aortic orifice, when it may be troublesome. In this case we may assume that the cardiac plexus (which closely embraces the root of the aorta and adjacent parts) is irritated either by dilatation of the vessel or by implication of the nervefilaments by extension of inflammatory or degenerative mischief from the aorta.

The term *angina pectoris* is applied to especially severe paroxysms of pain, accompanied by an awful sense of impending death, not infrequently fatal, occurring for the most part after the age of 40, and generally associated with degenerative disease of the root of the aorta, which sometimes extends into the coronary arteries. No hard and fast line, however, can be drawn between such cases and those previously mentioned. The immediate pathological cause—neuritis or neuralgia of the cardiac plexus—is probably the same, only it is more severe in angina, the associated structural disease of the heart and vessels is generally more serious, and the condition of the system is less able to withstand the shock of an attack with impunity. Further, in severe cases, the shock radiates into various parts of the nervous system, and amongst others, to the vaso-motor centre in the medulla, producing thereby spasmodic contraction of the peripheral arterioles, which by obstructing the circulation exaggerates the cardiac distress.

The symptoms of angina present every degree of severity, but in a typical case, the pain comes on suddenly in the chest and radiates into the arms. The face and body become suddenly pale, cold, and bedewed with sweat. The countenance is expressive of the most intense anxiety, and dread of impending death. The breathing is shallow, frequent, and often distressing, but not really obstructed. The pulse is usually small and thready. At the height of an attack, syncope or general convulsions may supervene. The attack continues "from a few seconds to many hours." Sometimes there is only one attack; or it recurs at longer or shorter intervals, each becoming more severe than the last. The disease in its typical form is generally fatal sooner or later.

The *treatment* in the milder cases of cardiac pain should be addressed mainly to the removal of the general or toxic conditions on which the pain appears to depend. Temporary relief may be afforded by the use of diffusible stimulants and antispasmodics. For persistent use, no drug is of greater value than arsenic, taken regularly for a long period.

In true angina nothing succeeds better than the inhalation of nitrite of amyl. This drug relaxes the peripheral arterioles, lowers the blood-pressure, and relieves the heart. Nitro-glycerine (4 or 5 drops of a one per cent. solution for a dose), and hypodermic injection of morphia are also useful for the same purpose, and act much in the same way. Hot applications to the chest, faradisation of the cardiac region, with internal administration of diffusible stimulants, belladonna, and small doses of opium have been recommended. During the intervals the indications for treatment are similar to those of chronic valvular disease.

GRAVES' DISEASE.

Graves' disease, or exophthalmic goitre is a neurosis which is characterised by palpitation, prominence of the eyeballs, *(exophthalmos)*, and enlargement of the thyroid gland *(goître)*. It is much more common in women than in men, especially in those of a nervous temperament. As to its precise nature we know very little; but it is usual to regard it as a neurosis of the sympathetic system which causes relaxation of the arteries, and excited action of the heart.

The *symptoms* may come on suddenly, but more often the onset is gradual. The subjects of it are usually nervous, emotional, irritable persons, with irregular appetite and digestion, and more or less anæmic. The first complaint is generally of *palpitation* with extreme frequency of the pulse, which is exaggerated by the least excitement. If organic disease of the heart be present, it is only coincidental. After a while *prominence of the eyeballs* is noticed, and this gradually increases until at last the eyelids are no longer competent to cover the eyes completely. Under such circumstances the eyes are apt to become inflamed. The *enlargement of the thyroid* is uniform and develops very slowly. The gland is soft, elastic, and pulsatile ; and a systolic thrill or murmur may be often heard over it. In some cases the exophthalmos or goitre may never take place. Although death is a rare termination, yet complete recovery is quite exceptional, and the course is excessively chronic.

Treatment.—The general health requires careful attention, and any condition which tends to weaken the system must be removed where possible. Medicinally, quinine or iron, in combination with digitalis, belladonna, or ergot is recommended. In one or two cases I have seen striking benefit result from a course of iron with arsenic, and also from daily galvanisation of the nerves of the neck.

MALFORMATION OF THE HEART.

A very brief allusion to congenital malformations will be sufficient for our present purpose. The most important of these are, persistence of the foramen ovale, with or without stenosis of the orifice of the pulmonary artery ; stenosis or obliteration of the aortic orifice ; and a persistence of a communication between the two ventricles. In any case the effects are very similar, retardation of the blood-current, and cyanosis. It is a noteworthy fact that in some cases symptoms are altogether absent; and in others are delayed for a variable period after birth. Diagnosis of the exact condition cannot generally be made with certainty during life.

Cyanosis implies more or less marked blueness or lividity of the skin. Two factors are necessary for its production—venous and capillary engorgement, and mal-aeration of the blood. Though in an extreme and persistent form it is associated with cardiac malformation, yet it is of common occurrence in chronic bronchitis, emphysema, cholera, valvular disease of the heart, or obstruction in the air-passages. The colour is most marked about the cheeks, lips, nose, ears, and the ends of the fingers and toes. The surface of the body is cold, and the finger-ends are often clubbed in chronic cases. The patient is dull and languid, and disinclined for exertion, mental or physical. He is also liable to attacks of oppression of breathing, palpitation, and syncope.

ACUTE PERICARDITIS.

Acute Pericarditis occurs most frequently as a complication of acute rheumatism or Bright's disease; or from extension of adjoining inflammation; or secondary to new growths such as cancer or tubercle.

The *anatomical changes* resemble, on the whole, those met with in inflammation of the pleura (*see* Pleurisy). The effusion is sero-fibrinous as a rule; and when bloody, is generally the result of new growths. Complete resolution is rare, and more or less extensive adhesion and agglutination usually persist.

Symptoms.—Symptoms are entirely absent in some cases; while in others they are masked by symptoms belonging to the primary affection. In a typical case, however, the attack is ushered in by pain and oppression over the region of the heart, palpitation, dyspnœa, and fever. The pain varies much in intensity, and usually subsides on the occurrence of effusion. It may be accompanied with tenderness on localised pressure. The amount of fever varies in proportion to the severity of the inflammation, and is attended with the usual febrile symptoms. The face wears an anxious expression, and the decubitus is generally dorsal. The breathing is accelerated and restrained at the outset, and becomes distressing and difficult on the occurrence of effusion, sometimes amounting to orthopnœa. It is accompanied with a dry irritable cough. The pulse is frequent, out of proportion to the breathing; and is liable to sudden variations from slight causes. There is usually more or less nervous disturbance indicated by headache, sleeplessness, and sometimes delirium or convulsions; and occasionally the nervous symptoms are so prominent as altogether to mask the local affection.

The *physical signs* during the *dry stage* consist of diffused impulse with excited action of the heart. In the *stage of exudation* we meet in addition with pericardial friction-sounds, and occasionally with friction-fremitus. In the *stage of effusion* the precordia is often bulged; the apex-beat (if it can be felt) is displaced upwards, undulating and irregular in force and rhythm, but more frequently it cannot be felt. The area of cardiac dulness is increased, first in an *upward* direction, then laterally. The shape of the dull area is very characteristic, being triangular, the base resting on the diaphragm. The friction-sounds often persist throughout the effusion, but generally become weaker, and sometimes disappear altogether. The heart-sounds are feeble, distant, and muffled, and are heard better at the base than at the apex of the heart. Adjacent organs are displaced, viz., the lungs, liver, and diaphragm. In the *stage of absorption*, the cardiac impulse re-appears, the apex-beat falls, the friction returns, and the area of dulness diminishes in extent.

The distinctions between endocardial murmurs and pericardial friction-sounds have already been given when dealing with the diagnosis of chronic valvular affections of the heart. The increased area of dulness may be distinguished from that due to cardiac enlargement by the shape of the area, the feeble or absent apex-beat—which is never displaced downwards and outwards in simple pericardial effusion—and the usual absence of mechanical troubles in the circulation.

The *prognosis* of acute pericarditis varies according to circumstances. Uncomplicated rheumatic pericarditis almost always ends favourably after a duration of from one to three weeks; but cases which are secondary to Bright's disease are generally fatal. Much depends on the presence or absence of complications, especially endo- or myo-carditis. The latter is particularly dangerous as tending to cardiac failure.

The *treatment* in the early stages consists of rest, with warm and moist applications to the chest. The pain when severe may be relieved by a hypodermic injection of morphia, or the application of a few leeches to the precordia. No medicinal remedies are specially indicated beyond those employed for the disease to which pericarditis is secondary. Plenty of nourishment is required; and sometimes alcoholic stimulants also, especially in the later stages. In exceptional cases the effusion may be drawn off by the operation of paracentesis, in order to relieve urgent symptoms. During convalescence tonics are called for.

Adherent Pericardium.—From a pathological point of view recovery from acute pericarditis is seldom complete, followed as it is by more or less permanent adhesion of the pericardial surfaces. When the adhesions are partial, and in the absence of endo- or myo-carditic complications, symptoms may never occur. More often the adhesions are sufficient to prevent the complete systolic emptying of the heart cavities; and, in accordance with rules already laid down, more or less dilatation and hypertrophy follow. It is clear also that these results will be aggravated by disease of the cardiac substance or valves. Such considerations sufficiently indicate the kind of symptoms that may be expected after severe pericarditis. The most important physical sign of adherent pericardium is systolic depression of the precordia in the neighbourhood of the apex beat, followed in many cases by a perceptible diastolic rebound, in virtue of the natural elasticity of the chest-walls. It is not easy, however, to diagnose adherent pericardium with absolute certainty.

Sub-Section II.

DISEASES OF THE VESSELS.

INFLAMMATION AND DEGENERATION.

Acute inflammation of the arteries is rare, and when it occurs, is almost always secondary. In the veins it is more common (phlebitis), especially in connection with pyæmic infection. Thrombosis is the invariable result of acute vascular inflammation; and, on the other hand, septic thrombosis may set up secondary phlebitis. The anatomical changes are analogous to those of acute inflammation generally.

Chronic arteritis is of common occurrence, especially in connection with gout, interstitial nephritis, persistent over-exertion, and syphilis, and is characterised by fibro-nuclear thickening of the vascular walls, particularly affecting the intima (endarteritis). The lumen of the vessels is encroached upon, and when small arteries are affected there is considerable interference with the circulation through them, and thrombosis is apt to occur. Obliterative thickening of the vasa-vasorum is probably an important factor in the initial causation of atheroma. From interference of the blood-supply, specific areas of arterial wall exhibit cloudy swelling, followed by fatty degeneration. The etiology of chronic general arteritis is only very imperfectly understood; but it appears to be primarily related to some persistent alteration in the character of the blood.

Chronic phlebitis most frequently results from venous thrombosis, or from chronic inflammation of adjacent tissues.

Atheroma in its earliest stages consists of patches of fibro-nuclear thickening affecting the deeper layers of the intima. Flattened prominences are thus formed on the lining of the artery, of an opaque whitish colour; afterwards becoming yellow, as the patch undergoes fatty degeneration. The patches are hard, and cut like cartilage. The subsequent progress varies according to circumstances. If the process be rapid, softening of the degenerative material takes place, and a pseudo-abscess, or atheromatous ulcer, is formed. In the more chronic forms, a caseous mass remains imbedded in the wall of the artery, and calcification frequently follows. The arch of the aorta, and arteries of the brain, are specially liable to atheroma, but no artery is exempt from it.

Atheroma diminishes the calibre of a vessel, destroys its elasticity, interferes with its contractility, and renders it less resistant and more fragile. These faults respectively create a liability to thrombosis, difficult capillary circulation, interference with the local regulation of blood-supply, aneurism, and hæmorrhage. Atheroma of superficial arteries can readily be made out by manipulation. When deeper vessels are affected, the condition can only be indirectly inferred.

Fatty degeneration of the intima is one of the many indications of chronic impaired health and senile decay. It must not be confused with atheroma. It may affect any of the arteries, and occurs in the form of yellow opaque slightly elevated patches on the lining membrane of the vessel. The cells of the intima are alone affected in the first instance, though later on the process may extend beyond this limit. Occasionally it leads to superficial erosion of the surface.

Calcareous infiltration of the arteries has already been referred to as secondary to atheroma, but it sometimes occurs as an independent condition, particularly in the aged. The muscular fibre-cells of the middle coat are chiefly affected. Small calcareous plates are thus formed; and in time the vessel becomes narrowed, rigid, and brittle. It frequently co-exists with atheroma in other parts of the arterial system.

AORTIC ANEURISM.

An aneurism is an abrupt dilatation of a vessel, with or without rupture of its coats. It generally occurs as a result of atheroma, or primary fatty degeneration of the arterial walls, which thus become weakened, and finally give way under the centrifugal pressure of the blood within. Aneurism is rare in the young; and is much more common in men than in women, especially in those given to violent exertion.

The student is referred to larger text-books on the subject for a full description of all the varieties of aneurism. Suffice it to say here that the majority of aortic aneurisms are irregularly saccular, involving the whole or a part of the circumference of the vessel; and in the latter case communicating with the artery by an opening of variable size and shape. The arterial coats, as a rule, can only be traced a short distance into the walls of the aneurism, which are formed for the most part by modification of the structures with which it is in contact. As the tumour grows, all soft parts are first compressed by it, then altered and finally blended inseparably with its walls. Hard, resistant structures, such as bone, are gradually laid bare, eroded, and absorbed. The cavity of the aneurism almost always contains more or less fibrin deposit in layers upon its inner surface in various stages of consolidation. The size of the tumour varies widely. Its effects mainly depend on pressure and displacement of the structures and organs around it; while as to its results, it generally proves fatal by rupture sooner or later. Sometimes it undergoes a spontaneous cure by coagulation of its contents, and sometimes its progress may be arrested by artificially bringing about a similar result; but such cases are quite exceptional.

Symptoms.—Sometimes no symptoms occur during life, or if present are often so obscure as to render a diagnosis impossible. In such cases, sudden death from rupture of the sac may first reveal the nature of the case.

Thoracic Aortic Aneurism.—As the symptoms mainly depend upon pressure of the surrounding parts, they necessarily vary according to the situation of the sac. It will be convenient to consider them *en bloc*.

Pressure on the air-passages.—The trachea may be displaced laterally, or its calibre may be diminished. In the latter case there is stridor, dyspnœa, and cough, all of which are increased by excitement or exertion. The cough is paroxysmal and suffocative, and is attended with scanty viscid expectoration. Pressure on a large bronchus is apt to be attended with imperfect expansion and feeble respiration on the same side, and may ultimately induce congestion or chronic pneumonia.

Pressure on the œsophagus is indicated by the ordinary symptoms of œsophageal stricture.

Pressure on the nerves.—Compression of the intercostal nerves or of the brachial plexus causes gnawing, burning, or shooting pains in the parts to which they are distributed. Compression of the trunk of the vagus nerve is sometimes credited with the production of dyspnœa, and inflammatory changes in the lungs. Pressure on either recurrent nerve is attended with paralysis of the laryngeal muscles, causing dysphonia or aphonia. Pressure on the sympathetic nerve gives rise to inequality of the pupils.

Pressure on the vessels.—Compression of the left subclavian or innominate artery may diminish, obliterate, or retard the pulse at one or other wrist. Pressure on the vena cava causes dilatation and engorgement of the veins above, and sometimes dropsical swelling of the neck; while pressure on the left innominate vein leads to distension of the veins on the upper half of the left chest-front.

Physical signs.—(1) Various degrees of local bulging and expansile pulsation may be observed; the site depending on the position of the aneurism. In the ascending part of the arch, it usually occurs to the right of the sternum in the second intercostal space; in the transverse part, at the upper end of the sternum; in the descending aorta, to the left of the dorsal spine. When the aneurism is deep-seated, as in the concavity of the arch, both external bulging and pulsation may be absent. (2) Dulness, with increased resistance on percussion, is obtained over the tumour. (3) On auscultation, systolic or diastolic "murmurs" may be heard, but a single diastolic murmur is very rare. These murmurs require careful distinction from those due to valvular disease of the heart, which so often co-exists with aneurism. (4) A thrill may sometimes be felt. Cardiac hypertrophy is rare in thoracic aneurism unless complicated with valvular lesions.

The ascending part of the aortic arch is the most common seat of thoracic aneurism, but any part of the vessel may be affected. Death is the usual termination, and may be due either to rupture followed by hæmorrhage, to asthenia, or to secondary complications.

The most common affections from which thoracic aneurism must be distinguished are—aortic valvular disease with cardiac hypertrophy and dilatation, mediastinal tumours, and abscesses or new growths in the thoracic wall.

Abdominal Aneurism may occur in connection with the abdominal aorta, or one of its large branches. It may usually be recognised as a pulsating expansile tumour in the abdomen, of rounded shape and very variable size. Like aneurism elsewhere, it causes displacement, compression, or erosion of the parts with which it comes in contact. Among the more common symptoms are pain from compression or tension of the nerves; persistent vomiting from pressure on the stomach or bowels; œdema of the lower half of the body from pressure on the vena cava; and jaundice from pressure on the bile ducts. The aneurism may ultimately burst into the peritoneal cavity, or one of the hollow viscera.

The affections most liable to be mistaken for abdominal aneurism are—undue pulsation of the aorta in nervous emaciated subjects, and solid tumours with pulsation communicated from the aorta.

The *treatment* of aneurisms (thoracic and abdominal) is very unsatisfactory. Rest should be strictly enjoined; and only a limited supply of food allowed. To relieve the pain, hot fomentations, lead and opium lotions, or hypodermic injections of morphia may be used. Sedatives, such as chloral, or small doses of opium, are often of value. Medicinally, iodide of potassium in full doses, acetate of lead, and digitalis (?) have been recommended. The bowels require attention. Of operative measures galvano-puncture is the most promising, but with regard to the indications for its use, and the mode of operation, reference must be made to special works on the subject.

Sub-Section III.

MEDIASTINAL TUMOURS.

Primary new growths in the mediastinum are not uncommon, and originate for the most part in the lymphatic glands of the posterior mediastinum, or in the connective tissue around. They consist sometimes of cancer, but perhaps more frequently of sarcoma or lymphadenoma. Increasing in size they implicate the surrounding parts either by extension of the morbid process into their structure, or by exerting pressure upon them and thus disturbing their functions.

As a rule, a mediastinal tumour can only be diagnosed with certainty when it has attained some considerable size. Speaking generally, the most characteristic features consist of the pressure-signs already described under the head of thoracic aneurism, together with such manifestations of general disturbance as may be peculiar to the growth in question. The diagnosis is further assisted, if we find on physical examination that the mediastinal dulness is increased in breadth, that the heart is displaced, that the glands of the neck are enlarged, or that the original disease is extending to the lungs or pleura, or forming secondary deposits elsewhere. Solid mediastinal tumours differ from aneurism in the absence of expansile pulsation and "blood-murmurs."

The *treatment* is purely palliative.

SECTION VI.

DISEASES OF THE ALIMENTARY SYSTEM.

SUB-SECTION I.

DISEASES OF THE MOUTH AND THROAT.

STOMATITIS.

INFLAMMATION of the mouth chiefly occurs in young children, and presents three varieties.

Catarrhal stomatitis.—In its simpler forms, it is characterised by general redness, swelling, and increased secretion of the mucous membrane, together with a good deal of local soreness. In more severe cases, it is attended with the formation of vesicles which rupture and discharge their contents, leaving a rounded ulcer, with well-defined margins, and a grayish floor. There is always more or less disturbance of the general health, with feverishness, and digestive derangement. This variety is commonly associated with difficult dentition, gastro-intestinal irritation, or certain specific fevers.

Ulcerative stomatitis consists of yellowish patches or spots of diphtheritic inflammation on the mucous membrane of the gums, lips, cheek, or tongue, followed by more or less extensive unhealthy-looking ulcers, with raised and congested margins which bleed readily when touched. If properly treated they generally heal quickly. It is common in unhealthy cachectic children who are badly fed and tended; and as a result of mercurialism it may occur at any age. There is considerable soreness, especially on chewing food; the breath is fœtid, and the neighbouring glands are enlarged. The general condition is characterised by marked depression and debility, with more or less fever. Ulcerative stomatitis is undoubtedly due to some specific cause, and is sometimes epidemic.

Gangrenous stomatitis or *noma* is a rare affection which is almost confined to children of weak and delicate habit. The first indication of mischief is fœtor of the breath, attended with the formation of a hard swelling in one cheek. The skin outside is red and shining, and on the mucous membrane within is an unhealthy sloughing ulcer. The inflammation includes the whole thickness of the cheek, then involves the gums and loosens the teeth; and if not checked, the bones are laid bare, the soft parts become gangrenous, and extensive destruction follows. If properly treated, healing with cicatrisation usually takes place, but sometimes it is directly or indirectly fatal. Strangely enough, there is but little fever, and the constitutional disturbance is trivial in comparison with the local mischief.

Treatment.—In all cases, the general state of health, the mode of life, and the hygienic surroundings require careful consideration. The food should be simple, fluid, and nutritious. The bowels should be carefully regulated. Medicinally, antacids, or rhubarb with soda or magnesia, are often of value, but the drug which is useful above all others is chlorate of potash, to which a little tincture of steel may be added with advantage. Frequent local application, with a brush, of chlorate of potash or borax lotions, or of equal parts of glycerine of borax and the B.P. solution of perchloride of mercury is beneficial; and obstinate ulcers may be touched with nitrate of silver. In *noma* the ulcer should be freely touched with strong nitric acid, followed by the use of antiseptic washes for the mouth, and external application of hot fomentations or poultices.

THRUSH.

Thrush is a term applied to an inflammatory condition of the buccal mucous membrane associated with the growth of a fungus known as *oïdium albicans*. It is a common disorder of children in connection with digestive derangements, but it is sometimes met with in adults. From the above fungoid growth, the tongue and mucous membrane of the mouth become covered with flaky white spots and patches. The mouth is hot and sore, the saliva dribbles away, and in infants, diarrhœa with acid green stools often co-exists, causing inflammation of the skin of the nates. Though it runs a tedious course if neglected, it is not in itself a dangerous disorder, and is usually very readily cured. In adults it is often a sign of ill-omen and impending death after long exhausting disease.

The *treatment* is essentially that already recommended for other forms of stomatitis.

GLOSSITIS.

INFLAMMATION of the whole thickness of the tongue is not a common affection, and is generally due to injury, stings, or mercurial poisoning. Sometimes, however, it is idiopathic.

The tongue becomes swollen, stiff, and incapable of executing its normal movements. It fills the mouth and often projects considerably beyond the teeth in front. There is a good deal of pain; while mastication, articulation, and swallowing become difficult or impossible. Sometimes suppuration occurs. It generally subsides spontaneously in the course of about a week; but now and then it proves fatal by extension of the inflammation and œdema to the throat, causing suffocation.

As to *treatment*, ice should be sucked, and the mouth should be frequently washed out with chlorate of potash lotion; the food should be liquid and nutritious, and if it cannot be swallowed, should be administered *per rectum*. In some cases, longitudinal free incisions of the dorsum of the tongue are required; and if suffocation threaten, tracheotomy may be necessary. If an abscess form, of course it should be opened.

PAROTITIS.

Parotitis or inflammation of the parotid gland occurs in two forms—(1) primary parotitis, and (2) symptomatic parotitis.

Primary parotitis or "mumps" is a specific infectious disease, and almost always occurs in an *epidemic* form. It is most common in the young. Although the change in the glands partakes of the nature of inflammation, there is no tendency to suppuration. Some amount of febrile disturbance precedes or occurs simultaneously with the glandular affection. The neighbourhood of the parotid swells and becomes painful, hot, and tender, thus rendering all movements of the jaw difficult and painful. Towards the end of a week the inflammation subsides, but in the meantime the opposite gland is apt to become affected in like manner. Not infrequently the inflammation leaves the parotid and flies to the testicles in males, or to the ovaries or mammary gland in females. This peculiar transference of symptoms is termed "*metastasis.*"

The *treatment* is exceedingly simple. Rest indoors for a day or two, a little mild saline aperient, together with the application of hot poultices or fomentations to the affected part, include all that is generally required.

Symptomatic parotitis or parotid bubo differs from the preceding form, in the fact that it is not specific or epidemic, and in its proneness to suppurate. It is generally met with as a sequela of typhus, typhoid, or some other specific fever, and is a complication of unfavourable import. The neighbourhood of the gland is swollen and boggy; and as suppuration advances, the skin becomes red, and fluctuates on palpation. The pus is ultimately discharged externally, or into the throat, or external auditory meatus, sometimes burrowing in the surrounding tissues. The general symptoms are of a low adynamic type.

By way of *treatment* hot fomentations or poultices should be employed, and when pus has formed it should at once be liberated by incision. A stimulant and supporting plan of treatment is necessary from the outset.

PTYALISM—SALIVATION.

ALTHOUGH strictly speaking salivation is only a symptom, it is one of sufficient importance to require separate notice. Secretory activity is always dependent on nervous stimuli brought to the gland from some peripheral source; in a word, it is essentially a reflex process. The causes of increased secretion of saliva will therefore depend either upon undue excitability of the salivary nervous centre, or on some abnormal peripheral irritation, or on both of these conditions together. Teething, stomatitis, facial neuralgia, pregnancy, uterine or ovarian derangement, and worms, are thus common exciting causes. Mercurial salivation comes into the same category, since it is due to the irritation and inflammation of the buccal mucous membrane which is set up by the prolonged use of mercury. Infancy, hysteria, and an emotional temperament rank as predisposing causes. Cases of ptyalism sometimes occur, *e.g.*, in the course of certain acute fevers, in which the mechanism is not clear. Increased secretion of saliva should be carefully distinguished from the constant dribbling which is so often met with in facial paralysis, general paralysis of the insane, and glosso-labial palsy, due to a difficulty in retaining the saliva in the mouth, or in swallowing it.

The *treatment* primarily consists in the removal of the cause wherever this possible. Mouth-washes of alum, chlorate of potash, or borax, are useful; and in many cases great relief is experienced by the internal or hypodermic employment of belladonna, atropine, or even morphia.

TONSILLITIS.

Inflammation of the tonsils is a common occurrence in the course of ordinary or mercurial stomatitis, measles, scarlet fever, diphtheria, and syphilis, but the present description applies exclusively to tonsillitis as a more or less independent affection. It is most common in the young, is peculiarly apt to recur, and in some instances it is distinctly epidemic in character. There is also a curious liability to tonsillitis in those subject to acute rheumatism. Exposure to cold or damp appears to be a frequent exciting cause.

It is convenient to distinguish two forms of tonsillitis.

1. *Catarrhal tonsillitis* consists of a superficial catarrh of the tonsils, soft palate, uvula, and upper part of the pharynx, with congestion and moderate enlargement of the tonsils, constituting the common "sore" or "relaxed throat." In such cases there is slight febrile disturbance accompanied with irritation, soreness, or pain in the throat, and discomfort on swallowing. The secretion is scanty at first, but afterwards more abundant. The voice is nasal, and articulation is often difficult. The above symptoms usually subside altogether in the course of a few days.

2. In *parenchymatous tonsillitis* or *quinsy*, the inflammation is more severe, and involves the tonsils as a *whole*. The inflammation may affect one or both tonsils, but

when both are involved, the affection generally begins in the second as that of the first is subsiding. The tonsils become greatly enlarged and congested. Its follicles become filled with yellowish coloured plugs consisting of accumulated and altered epithelium. The soft palate and fauces are red, swollen, and more or less displaced by the enlarged tonsils, to which an elongated and œdematous uvula often clings. The tissue of the tonsil is very apt to undergo suppuration. The disorder is ushered in with chilliness or an actual rigor, quickly followed by high fever (102°-105°), and the usual signs of febrile disturbance. Simultaneously, or soon afterwards, attention is attracted to the throat, which becomes very sore and painful, especially on attempting to swallow or on separating the jaws. When fluids are taken they are apt to regurgitate through the nose. On examination of the throat at this stage the appearances are such as have already been described. The salivary secretion is much increased and often dribbles from the mouth, the voice is nasal and characteristic, the hearing is impaired, and the glands at the angle of the jaw are always more or less tender and swollen. There is generally considerable depression, the tongue is furred, the appetite is lost, and the bowels are confined. If both tonsils are simultaneously affected, the swelling of the throat may cause great difficulty in breathing.

It is a noteworthy fact that the urine often contains albumen, and sometimes in large quantity; but this condition disappears almost invariably with the other symptoms. In a few days the malady attains its height and then subsides either with or without suppuration. The prognosis is generally favourable.

The *treatment* of both forms may be considered together. Among the more important general measures are, rest in a warm room, sucking ice, hot fomentations to the throat, steam inhalations, and good nutritious food. Owing to the difficulty in swallowing, the nourishment taken is often inadequate. It should be given in the fluid form, in small quantities at a time, and frequently repeated. Alcoholic stimulants are usually indicated in moderation, even from the first. Medicinally, much may be done. The bowels should at the onset be freely opened by a saline aperient, or a calomel and jalap powder. In the early stages drop or half-drop doses of tincture of aconite, with or without the same quantity of tincture of belladonna, given every hour, or oftener, are of great service and will sometimes cut short an impending attack when taken early enough. Salicylate of soda, chlorate of potash, and tincture of steel are also useful drugs. Dr. Ringer speaks very highly of hyd. c. cret. (gr. $\frac{1}{3}$ every hour) when suppuration has occurred, or when suffocation seems imminent from the swelling of the throat. To ensure rest at night, a moderate dose of Dover's powder, or chloral is sometimes required. During convalescence steel, quinine, and the like, together with an occasional local application of glycerine and tannin are of service. The abscess in the tonsil may be opened by incision in severe cases, but it is an operation which requires careful performance.

Chronic enlargement of the tonsils not infrequently succeeds acute tonsillitis, and occasions much annoyance by obstructing and irritating the throat, moreover, it is a common cause of deafness from occlusion of the Eustachian tube. If change of air, tonics, and local astringent applications fail to reduce the size of the tonsils, they should unhesitatingly be removed by the knife.

RETRO-PHARYNGEAL ABSCESS.

This affection, as its name implies, consists of suppuration behind the pharynx, between it and the vertebral column, and is generally connected with caries of the spine. A soft fluctuating tumour is thus formed at the back of the throat, which by its size may render swallowing and breathing difficult. It generally "breaks" into the throat. Its treatment is essentially surgical.

Sub-Section II.

DISEASES OF THE ŒSOPHAGUS.

The œsophagus may be *acutely* inflamed by the passage through it of hot, irritating, or corrosive matters; and also in connection with inflammation of adjacent parts (croup, diphtheria, &c.). Febrile disturbance, with pain along the œsophagus, which is increased on swallowing, form the most prominent symptoms.

The *chronic* affections of the œsophagus claim our consideration at greater length, and may be classified as follows:—

1. Functional disorders—(*a*) spasm, and (*b*) paralysis.
2. Organic diseases—(*a*) ulceration, and (*b*) new growths.

Two important secondary conditions may be added to the above, viz.:—stricture and dilatation of the œsophagus. The latter sometimes occurs independently of stricture.

Spasm of the œsophagus is most often met with in hysterical women who are the subjects of uterine or ovarian derangement; and it is usually of reflex origin.

Paralysis of the œsophagus is a common occurrence just before death, but it is also met with in bulbar paralysis, in paralysis of the insane, and in the last stages of progressive muscular atrophy.

Ulceration of the œsophagus.—Simple ulcers may arise from mechanical violence, or in the action of corrosive fluids. A perforating ulcer, similar to that met with in the stomach, is sometimes observed.

New growths of the œsophagus.—Of these cancer is by far the most common. It is usually primary, and is especially frequent at the level of the bifurcation of the trachea. Syphilitic growths followed by ulceration, and fibroid tumours are exceptionally met with.

Stricture of the œsophagus.—The causes of œsophageal stricture may be thus classified:—

Stricture of the Gullet may depend on	1. Simple nervous spasm.	
	2. Changes in walls	Thickening of walls from chronic catarrh. Cicatrisation of ulcers.
	3. New growths . .	Cancer. Fibroid polypi.
	4. External pressure	Aneurisms. Bronchocele. Enlarged glands. New growths.

The junction of the upper and middle thirds of the œsophagus is the most frequent seat of the stricture. Above the obstruction the tube is dilated and its walls are thickened; while below it is collapsed, and its walls are often thinned.

Excluding cases of simple spasm, the *symptoms* develop *gradually*, and essentially consist of difficulty in swallowing—with or without pain. The dysphagia is at first slight, and is only felt on attempting to swallow large solid morsels; but as the obstruction becomes more complete, both liquids and solids pass with difficulty. Before long, regurgitation of the food takes place, either as soon as it is swallowed, or after an interval, the length of which is in direct proportion to the low position of the stricture, and the amount of dilatation above it. A good deal of mucus is also regurgitated with the food, together with blood or pus if there be ulceration. Such regurgitation differs from stomach-vomiting by its easy spontaneous occurrence, the alkaline reaction of the ejecta, and the absence of any nausea. Pain or discomfort when present is generally referred to a point behind the manubrium sterni. Unable to take adequate nourishment, the patient becomes weak and emaciated (though probably retaining a good appetite), until finally he dies of starvation. The course and progress of any given case obviously depend on the nature of the condition causing the stricture.

General Diagnosis.—The use of the bougie will serve to distinguish organic stricture from simple spasm, paralysis and dilatation. Simple spasmodic stricture comes on suddenly, runs an irregular intermittent course, and generally occurs in those of an excitable nervous temperament. In paralysis, solids are more easily swallowed at first than liquids; but in the later stages, deglutition is impossible, and the saliva dribbles away from inability to swallow it. Dilatation in connection with stricture is indicated by the interval which elapses before regurgitation occurs. Hiccup also is a frequent symptom; and where saccular pouches exist, the breath is very fœtid. The history of the patient, the age, temperament, and the progress of the case should always be carefully enquired into. Advanced age, pain, cachetic appearance, and a rapid course point to cancerous stricture; while regurgitation of blood or pus usually points to ulceration of some kind (malignant or otherwise). The neck and thorax should also be carefully explored or bronchocele, aneurism, mediastinal and other tumours.

Treatment.—Functional obstruction may often be overcome by simply passing a bougie; but treatment should always be directed to the nervous condition on which it depends. In organic stricture the treatment is very unsatisfactory. Bougies should never be used except for diagnostic purposes (and then only with great caution) where there is any reason to suspect ulceration, but in simple fibroid stricture their systematic use is sometimes beneficial. When the strength fails from want of proper nourishment, nutrient enemata should be given. In some cases gastrotomy has been performed, but without much success.

Sub-Section III.

DISEASES OF THE STOMACH.

SYMPTOMATOLOGY.

It will in the first place be well to enumerate some of the more important *disturbances of function* arising from gastric disorder, with brief comments on their nature. Of these, *pain*, *flatulence*, *water-brash* or *pyrosis*, *heartburn*, *vomiting*, and *hæmatemesis*, are referable to the stomach itself.

1. *Pain* is altogether absent. Its seat may be in the stomach itself, deep-seated behind the sternum, between the shoulders, or reflected to some part on the left side.

It varies from mere uneasiness or a sense of oppression to an intense degree of severity—with or without *tenderness*. It may arise (*a*) from direct irritation of the stomach by flatulent distension, the products of fermentation, undigested food, &c.; (*b*) from organic disease of its walls; (*c*) from excess or alteration in the quality of its secretions; or (*d*) as a neuralgic affection (see Gastralgia). It is important to note that, as a rule, pain from functional disturbance is temporarily relieved by food, does not recur until some time after food, and is relieved by pressure; while pain from organic disease comes on soon after food, is not relieved until the latter is ejected, and is aggravated by pressure.

Flatulence may affect the stomach or bowel, and arises in one of two ways. Either it is the result of swallowing air, or of fermentation processes from delayed or altered digestion. In the former case the "flatus" consists of atmospheric air, the oxygen of which is more or less replaced by carbonic acid gas, as the result of respiratory changes effected through the membranes of alimentary canal. Swallowing movements are a common result of throat or stomach irritation in nervous subjects, and many cases of rapid distension of the stomach occurring independently of food may be thus explained. The air thus swallowed passes on into the bowel. Flatulence resulting from fermentation processes consists chiefly of carbonic acid gas, but there is often a variable amount of sulphuretted hydrogen or marsh gas.

3. *Water-brash* or *pyrosis* is a paroxysmal affection, rarely occurring before puberty, beginning with epigastric pain or discomfort, as a rule followed after an uncertain interval with the discharge of fluid without nausea or effort. It may and often does lead on to actual vomiting of the contents of the stomach. The

fluid has all the characters of clear saliva—provided it is not accidentally mixed with vomited matter. From a careful recent study of the subject,* I regard water-brash as paroxysmal salivation, dependent upon reflex irritation, sometimes taking its origin in disease of the stomach, sometimes in some irritating article of food as oatmeal, sometimes in disorder of some distant part as the uterus or ovaries. General nervous excitability is also often an important factor in its causation. The amount of fluid which is discharged varies from a mouthful to half a pint or more.

4. *Heartburn* is the term applied to a disagreeable and peculiar burning sensation along the entire gullet, associated with the regurgitation of an acrid fluid from the stomach, which in all probability consists of butyric acid, a product of abnormal digestion.

5. *Vomiting* may be conveniently classified according to its causes. (1) *Stomachic* vomiting—which depends on reflex irritation and is due to functional or organic disease of the stomach. (2) *Nervous* vomiting—which may be divided into two minor groups:—(*a*) *Reflex* nervous vomiting from peripheral irritation occurring elsewhere than at the stomach, as in hepatic and renal colic, strangulated hernia, ovarian and uterine disorders. Vomiting also from meningeal irritation, giddiness, and certain cerebral tumours is, from a physiological point of view, truly reflex. (*b*) *Central* nervous vomiting is exclusively due to *direct* irritation of the vomiting-centre)presumed to exist in the medulla) from certain brain diseases, toxic agents (as tartar-emetic), altered conditions of blood (as in uræmia), onset of acute fevers, or extreme depression.

Stomachic vomiting is usually preceded by nausea, attended with a furred tongue and other indications of gastric disorder, and usually gives relief to any nausea, vertigo or headache which may precede it. In *nervous* vomiting, nausea is usually absent, the tongue is clean, and symptoms of brain disease, if present, are not relieved by it.

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* *Vide* "Practitioner," May, 1890.

Vomited matter varies much in quantity, and may consist of food either unaltered, or partially digested; or it may be frothy and present evidences of fermentation; or it may contain bile, mucus, blood, or pus. Its reaction is generally acid. Fæcal vomiting only occurs as the result of intestinal obstruction, or of some direct communication between the stomach and colon. Examined *microscopically*, peculiar little square or oblong vegetable organisms like woolpacks, called *sarcinæ*, may be found, especially in connection with pyloric obstruction.

6. *Hæmatemesis* or blood-vomiting is characteristic of bleeding into the stomach, only excepting those cases in which blood from the nose, mouth, or air-passages, has been previously swallowed. This may arise from (*a*) external injury; (*b*) acute congestion due to acute inflammation, or suppressed menstruation; (*c*) mechanical congestion from *cirrhosis of the liver*, pressure on the portal vein, or long-standing disease of the heart or lungs; *(d)* erosion of vessels, from *ulcer or cancer;* (*e*) disordered state of the blood, as in scurvy, or yellow fever; and *(f)* in rare cases, from the bursting of an aneurism into the stomach.

Symptoms.—Small hæmorrhages which are not followed by vomiting may pass unnoticed. In other cases, small quantities of blood in the vomit may be the sole indication that hæmorrhage has occurred. When the bleeding is sudden and profuse, there is at first a sense of sinking at the epigastrium, sudden pallor, a small frequent pulse, and faintness, which is followed by nausea, and the ejection of dark-coloured partly clotted blood. The next stool contains blackened blood, and for some days after, the motions may be black and "tarry." When the hæmorrhage is scanty, the blood forms small dark-coloured granular clots, closely resembling "coffee-grounds." Though serious prostration may persist for a considerable period, a single attack is seldom fatal.

Hæmatemesis may be distinguished from hæmoptysis by (*a*) the blackish colour and acid reaction of the blood; (*b*) frequent mixture with food; (*c*) the nausea which usually precedes it; (*d*) expulsion by vomiting; (*e*) the evidence of blood in the subsequent stools; and (*f*) by symptoms indicating gastric disorder.

We may now briefly consider those *disturbances of function* which are *secondary* to the disorder of the stomach.

1. *Digestive system.*—The *tongue* may be variously affected; thus it may be red and irritable looking, or covered with white or yellow fur; or broad, flabby, and pale; or fissured; or patchy. The *appetite* is impaired or lost (*anorexia*). *Thirst* is common. The *bowels* act irregularly, and the appearance of the *stools* is altered.

2. *Nervous system.*—Weakness, weariness, troubled sleep, headache, depression, irritability, loss of memory, dimness of sight, vertigo, and hypochondriasis.

3. *Respiratory system.*—Asthmatic symptoms and dry cough.

4. *Circulatory system.*—Palpitation, and intermittent action of the heart.

5. *Urinary system.*—The urine may be altered in quantity and quality. Sometimes it is very acid, and deposits urates, uric acid, oxalate of lime, &c.; in other cases it is alkaline after meals, and deposits amorphous phosphates.

6. The *skin* may be harsh, or greasy, or hot and dry; and is liable to eruptions—especially in children.

7. The *general nutrition* is impaired, as shown by emaciation and anæmia.

FUNCTIONAL DISEASES OF THE STOMACH.

DYSPEPSIA.

THIS term is applied to those forms of indigestion which are not attended with structural changes of the stomach. Dyspepsia is proximately due to one or both of two conditions—(*a*) *defective secretion*, and (*b*) *impaired movements* of the stomach.

The *causes* of both may be taken together. 1. Defective quality of the food. 2. Excessive quantity of food. 3. General weakness from any cause. 4. Disordered blood-states. 5. Causes operating through the nervous system—mental and physical exhaustion, shock, narcotic drugs, &c.

SYMPTOMS.—The leading symptoms of the two forms may be thus contrasted.[*]

IMPAIRED MOTION.	DEFECTIVE SECRETION.
Uneasiness after meals.—A constant symptom; generally soon replaced by the sense of tension accompanying flatulence.	*Uneasiness after meals.*—Not infrequent, but soon merged in actual pain.
Flatulence.—This is the most characteristic symptom of impaired motion.	*Flatulence.*—Comparatively infrequent. Some of the worst cases in which pain after food and other symptoms are particularly severe, are entirely free from flatus. The tendency is to forms of fermentation in which gases are not evolved.

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[*] For this table I am indebted to a paper by Dr. Leared.

Gastric pain.—Infrequent, but occurs occasionally as a result of flatulence, and is peculiar in kind.	*Gastric pain.*—Variously described as sharp, shooting, dull or dragging, is the most characteristic symptom of defective secretion.
Constipation.—Almost always a marked symptom.	*Constipation.*—Not generally present, and the bowels are often relaxed.

In both there is loss of appetite. The tongue is broad, pale and flabby. The pulse is weak, soft, and compressible, and palpitation is common. There may be dyspnœa on exertion, and a dry cough. The complexion is pale. The urine is pale, depositing sometimes oxalates or phosphates. Nervous symptoms, as already enumerated, may be present; and the general nutrition suffers.

Gastralgia.

Gastralgia is a *chronic neuralgic affection* of the stomach especially frequent in hysterical females. It is also associated with general debility, mental strain or worry, shock, sedentary habits, gout and rheumatism, and abuse of stimulants and tea.

Symptoms.—*Pain* at the epigastrium, often very severe, relieved at first by food, and also by firm pressure. The *appetite is depraved* or lost; there is *frequent vomiting* especially in hysterical subjects; and the *bowels are very constipated.* Over and above these symptoms we meet with those of ordinary indigestion.

Spasm or Cramp of the Stomach.

This is an acute affection due to painful spasmodic contractions of the stomach, brought on by over-distension of the organ by food or flatus, by irritating ingesta, or by taking iced drinks in excess, especially when heated, or immediately after meals.

SYMPTOMS.—The patient tosses or rolls about with *severe pain* at the pit of the stomach, which comes on *suddenly*, is of a griping or twisting character, and is markedly relieved by firm pressure, or vomiting. It is often attended with prostration, clammy sweats, a small thready pulse, and palpitation. Some soreness over the stomach is apt to persist for a few days afterwards. In rare cases it may be fatal.

GENERAL TREATMENT OF FUNCTIONAL DISEASE.

Dietetic.—All indigestible food, as pork, veal, and salt meats, and rich made dishes should be avoided. Vegetables and fruits should only be taken sparingly, and pastry or sweets are especially harmful in acid forms of dyspepsia. In some cases an exclusively *milk diet* is necessary for a time, with the addition of barley water to prevent its coagulation into lumps. *Water* should be the habitual drink. Cocoa (free from fat) is often preferable to either tea or coffee. If the latter are taken at all, they should be *weak, cool, and well diluted with milk.* Rich or effervescent wines should be avoided, but light wine or ale is often useful in atonic dyspepsia.

Hygienic.—Exercise, fresh air, avoidance of hard mental or sedentary work, cold or tepid bathing, and warm clothing.

Medicinal.—For *undue acidity* with scanty and loaded urine, alkalies are useful, and are best taken three or four hours after a meal; in acidity with anæmia and debility, mineral acids are better. Vegetable bitters (of which calumba is the mildest), and strychnia or nux vomica are good *digestive tonics.* In *slow digestion* ipecacuanha (gr. $\frac{1}{2}$ before meals) may be tried. For *vomiting*, ice to suck, effervescents with hydrocyanic acid, bismuth, morphia (subcutaneously), liq. arsenicalis (♏ $\frac{1}{2}$-i. before food), nux vomica (♏ $\frac{1}{2}$-i. every few hours) or counter-irritation at epigastrium. For *flatulence*, warm carminatives, charcoal, bismuth, nux vomica, and alterative aperients. For *pain*, gr. v. of the carbonate of iron with aloes, minute doses of liq. arsenicalis, morphia (subcutaneous only), or bismuth. *Pepsine* (gr. iij.-x. taken at meal times) is very useful in some forms of weak digestion.

GASTRIC CATARRH.

Acute gastric catarrh is commonly caused by indigestible food (especially in children), irritant poisons, or alcoholic excess. It also occurs in connection with gout, or as a symptom of acute fevers.

The *anatomical changes* are, active congestion (leading sometimes to small extravasations), swelling, and occasionally superficial or follicular ulceration of the mucous membrane, especially in the neighbourhood of the pylorus; increased secretion of mucus; and infiltration of the membrane with young cells.

Symptoms.—There is often severe *pain* in the epigastrium, which is increased by food and is relieved by vomiting. *Tenderness* is always present; *nausea* and *vomiting* are also prominent symptoms; the vomit consisting of glairy mucus stained with bile. The *tongue* is foul, while the edges and tip are not unfrequently red and irritable-looking. The bowels are generally confined, and the urine is scanty and high-coloured.

The above symptoms set in with chilliness, followed by *fever* with restlessness, headache, and insomnia. In bad cases there may be great collapse, with cold clammy skin, a rapid weak pulse, and obstinate hiccup with some dyspnœa.

The symptoms, as described, usually subside after a variable time, but sometimes the disorder passes into a chronic form. In uncomplicated cases, the prognosis is generally favourable.

Treatment.—If due to poison or irritant matter still remaining in the stomach, this must be got rid of by an *emetic*, followed by a *mild purgative*. The *diet should be entirely liquid*, preferably consisting of milk well diluted with soda or seltzer-water, given in small quantities at a time. The *thirst* should be relieved by sucking small pieces of ice. *Stimulants* are rarely required except in threatening collapse; but champagne is recommended where the vomiting is persistent, and apparently due to sheer exhaustion. Medicinally, bismuth and hydrocyanic acid, or saline effervescents may be given to relieve the pain and allay excitement. In obstinate cases, the hypodermic injection of morphia is sometimes of valuable service. *Locally*, the application of poultices or fomentations to the abdomen, or of a few leeches to the epigastrium may give relief. The *convalescence* is slow and requires care.

CHRONIC GASTRIC CATARRH or irritative dyspepsia may occur as a sequel of the acute form, but more commonly it arises from severe or frequently recurring irritation by improper food or alcohol, from imperfect mastication, from portal congestion, or in connection with some organic disease of the stomach.

Anatomical changes.—Increased vascularity of the lining membrane, which is covered with thick tenacious mucus. The membrane itself is firmer and thicker than usual, from an increase of its interstitial tissue. Its gland-structures are also variously altered.

Symptoms.—These so closely resemble the symptoms of simple dyspepsia that it will suffice here to direct attention to the main points of difference, viz.:—greater amount of epigastric discomfort and tenderness; thirst; a large tongue indented by the teeth at its edges, and usually coated with a thick yellowish fur; greater frequence of acidity, heart-burn, water-brash, and vomiting; high coloured urine, depositing urates; and greater severity of the constitutional symptoms.

Treatment.—Successful treatment of these cases depends on the possibility of detecting and removing the source of irritation. As a rule, aperients are needed; those of the saline type assisted by mild mercurial alteratives suiting best. When vomiting is a prominent symptom, purgatives of any kind generally do harm. In the early and more urgent stage alkalies, bismuth, oxide of silver, with nux vomica or columba are indicated; but later on, quinine, iron, and the mineral acids are to be preferred. The same general principles must be observed, which have been described under the head of functional gastric disease.

ULCER OF THE STOMACH.

The simple or non-inflammatory ulcer of the stomach is due to the destructive action of the gastric juice upon localised areas of mucous membrane, the vitality of which has been impaired or arrested—usually as the result of sub-mucous extravasation or of thrombosis. It is very rare in children, but afterwards becomes more frequent as age advances.

Anatomical Appearances.—A simple gastric ulcer is round, from one-fourth to one-half an inch in diameter, with thin clean-cut margins, deepest in the centre like a shallow funnel, but varying in depth from mere destruction of the mucous membrane to complete perforation. It is usually single, and most commonly situated on the posterior wall near the pyloric end. Erosion of important vessels may occur, leading to serious hæmorrhage. As to its terminations—(*a*) it may cicatrise with or without puckering; or (*b*) *perforation* may occur, with escape of the contents of the stomach into the peritoneal cavity, unless this be prevented by *adhesions* with neighbouring parts.

The gastric mucous membrane elsewhere may exhibit changes indicative of chronic catarrh.

Symptoms.—Epigastric *pain* and *tenderness* are the most constant and important symptoms of gastric ulcer. The pain comes on shortly *after* food, and increases till the stomach is emptied by vomiting. Sometimes it extends to the back. The seat of tenderness coincides with that of the pain, and is strictly *localised*. *Vomiting* is a frequent symptom. Hæmatemesis occurs in about one-third of all cases; and though not so frequent as in cancer, is far more profuse. The bowels are generally constipated. Various dyspeptic symptoms are also met with, and in chronic cases the *general nutrition* suffers considerably.

The above symptoms vary somewhat according to the position of the ulcer, and in rare cases may be altogether latent.

The duration of gastric ulcer varies greatly. The disease terminates in one of three ways:—(1) Gradual recovery by cicatrisation, which is the rule; (2) sudden death from perforation, peritonitis, or from hæmorrhage; (3) gradual death from exhaustion. The impossibility of foreseeing the occurrence of hæmorrhage or perforation introduces an element of uncertainty in the prognosis of gastric ulcer.

Diagnosis.—Careful attention to the symptoms as enumerated will usually suffice to distinguish ulcer from gastralgia. The diagnosis from cancer will be given in the description of the latter.

Treatment.—To attain physiological rest for the stomach is of the very first importance; hence food should only be given *in small quantities* at one time, and in a *liquid* form. In grave cases it is necessary to administer nourishment for a time exclusively by nutrient injections *per rectum.* For *pain*—morphia should be given (subcutaneously by preference). *To empty the stomach of acid products of digestion*—the saline aperient mineral waters, notably Carlsbad, Pullna, and Hunyadi Janos waters may be periodically exhibited. For *vomiting*, liq. arsenicalis (dose of ♏ j.-ij., diluted), bismuth, prussic acid, creasote, or ice to suck. For *hæmatemesis*, ice, ergotin (hypodermically, gr. j.-ij.), gallic acid, turpentine, or acetate of lead and opium, &c. *Enemata* may be needed to relieve the *constipation.* After disappearance of active symptoms, tonics are of great service, especi- the carbonate or citrate of iron.

CANCER OF THE STOMACH.

The stomach is one of the most frequent seats of cancer. Hereditary tendency, advanced life, the male sex, and chronic irritation form the more important predisposing causes.

Anatomical Appearances.—Cancer commonly affects the orifices of the stomach, notably the pylorus. It extends transversely, and hence is very apt to lead to annular stricture with obstruction.

Scirrhus is the most frequent type, *encephaloid* coming next, and still more rarely *colloid.* The growth takes its origin in the epithelial cells of the mucosa, gradually involving the deeper structures in the course of its development. Sooner or later it generally breaks down, the surface is laid bare, and presents an irregular, ulcerous, and highly vascular sore. Cancer of the stomach is almost always primary, and is apt to involve neighbouring organs and structures, either by direct extension, or as a secondary deposit.

The stomach is variously affected, according to the form, extent, and progress of the growth. More often the pylorus is displaced downwards by its own weight, the stomach is dilated and its walls hypertrophied.

Symptoms.—There is almost constant *pain* at the epigastrium, gnawing or lancinating in quality, and radiating to the back and shoulders. It is aggravated by food, and relieved by vomiting, but in neither case so distinctly as in ulcer. *Tenderness* is almost invariable. Vomiting is seldom absent, especially if the orifices be obstructed. When the pylorus is constricted the vomit is brownish, frothy, contains *sarcinæ*, and is not ejected for some considerable time after food; while with obstruction of the cardia, food is returned almost immediately, and but little altered. *Hæmatemesis* is an early symptom and occurs in the majority of cases. The hæmorrhage is generally scanty; and as it oozes away, it becomes coagulated and blackened by the action of the gastric fluids, giving rise to a "coffee-ground" appearance in the vomit. The stools under such circumstances present indications of the presence of blood (*melæna*). Various dyspeptic symptoms are also met with.

The following physical signs may be observed, viz.. *epigastric fulness* with *increased resistance;* a *tumour* in the pyloric region (above and to the right of the umbilicus), which is usually hard, irregular, more or less fixed, and tender on pressure; and the *signs of dilatation of the stomach* (see below).

Constitutional disturbance is indicated by early and rapid emaciation, progressive debility, a dirty sallow complexion, an anxious countenance, and pinched features—constituting together what is called the cancerous cachexia.

The average *duration* is about one year, and it is always fatal. Death occurs from exhaustion, and is sometimes preceded by femoral thrombosis.

DIAGNOSIS.—The chief points which serve to distinguish *cancer* from *ulcer* of the stomach are—its non-occurrence in the young; hereditary taint; constancy of the pain; the greater diffusion of the tenderness; frequently recurring and scanty hæmatemesis; marked constitutional symptoms beginning early; and later on, the *presence of a tumour*, with the signs of pyloric obstruction and gastric dilatation.

TREATMENT can only be palliative, and in principle is similar to that already given for ulcer of the stomach. It is deserving of mention, that great relief, and sometimes for a long period, can often be given in the early stages, by confining the diet to artificially digested food. When there is marked pyloric obstruction and gastric dilatation, periodic washing-out of the stomach, by means of a syphon tube, is most beneficial.

PYLORIC OBSTRUCTION—GASTRIC DILATATION.

CANCER is the most common cause of pyloric stenosis, but it sometimes arises from sub-mucous induration and thickening, or a contracted cicatrix after ulcer. As a necessary effect of the obstruction, the stomach *dilates;* but dilatation may and does occur independently of pyloric stricture, as the result of nutritive changes in the stomach-wall combined with systematic distension from any cause. The chief symptom of obstruction is *vomiting*, occurring some hours (or at even much longer intervals), after food. The vomited matter is abundant, very acid, exhibits a thick frothy scum on standing, and contains *sarcinæ*. To these symptoms must be superadded the physical signs of dilatation of the stomach, viz.:—prominence of the left upper part of the abdomen, hollowness of the epigastrium, extension of the area of stomach resonance on percussion, and splashing sounds easily produced by succussion of the abdomen.

SUB-SECTION IV.

DISEASES OF THE INTESTINES.

BEFORE proceeding to describe the diseases proper to the intestines, four important symptomatic conditions must be briefly discussed.

I. DIARRHŒA.

Diarrhœa is proximately due either to unusual fluidity of the intestinal contents, or to increased peristaltic activity of the intestinal walls, or to both of the conditions combined. These results may be brought about in the following ways:—(1) Irritation, inflammation, ulceration or new growths; (2) mechanical congestion from obstruction of the portal circulation; (3) collateral congestion from the sudden arrest of profuse discharges, or of perspiration by chill; (4) as a symptom of some general illness of a septicæmic nature; (5) from increased intestinal irritability due to fear, anxiety or shock; and (6) as a result of specific disease such as cholera, typhoid, dysentery, and summer diarrhœa.

The *characters* of the stools vary greatly according to circumstances. They may be *(a) fæculent*, or contain no true fæcal matter; *(b) brown*, *green*, or *yellow*, due to various alterations of bile pigment; *(c) slimy*, due to admixture with mucus; *(d) serous; (e) bloody;* or *(f) lienteric* containing scarcely altered food. They may be alkaline or acid; in the latter case they are very apt to irritate the anus and surrounding parts. They may be passed easily, or with straining (*tenesmus*). Diarrhœa may also be associated with various digestive disturbances, griping (*tormina*), or borborygmi. If it continues for any considerable period it is attended with debility, wasting, or prostration. It may be added that diarrhœa with slimy stools always implies an inflammatory condition of the bowels; and when accompanied with *tenesmus*, the lower or larger intestine is always involved.

The *treatment* depends entirely on the cause. In some cases it is inadvisable to check it, as in *uræmic* diarrhœa. If due to irritating matter which still remains in the intestine, it is well to give a dose of castor oil in the first place. Under other circumstances it may be checked by opium, with or without such astringents as sulphuric acid, catechu, &c. The food should be light and unirritating.

Opium is specially indicated when there is undue irritability of the bowel and in most cases of inflammatory diarrhœa. In the simple catarrhal diarrhœa, bismuth, oxide of zinc, aromatic chalk, and charcoal are more useful. When there is evidence of fermentative disorder, and in the summer diarrhœa of infants carbolic acid (♏ j., for a dose), creasote, and liq. hydrarg. perchlor. (♏ v.-x.)—with or without opium as required—are beneficial.

II. Constipation.

Constipation may arise from—(1) intestinal obstruction (which will be separately considered); (2) diminished peristaltic action; or (3) undue dryness of intestinal contents. Independently of organic disease, constipation is a frequent result of sluggish action of the bowels, associated with a weak state of health, sedentary employment, want of exercise, anæmia, or habitual neglect of the calls of nature. Undue restriction of fluid ingesta is also a very common cause of constipation, especially in women. Constipation leads to loss of appetite, bad complexion, imperfect digestion. and mental depression. When fæces accumulate in large quantities, an abdominal tumour may form, recognizable by palpation. In certain cases, hard impacted fæces may set up irritation, inflammation, and even ulceration of the gut, or lead to diarrhœa.

Treatment.—1. Careful regulation of the diet in which it is often well to include bran bread, oatmeal and certain fruits. 2. A full supply of fluid ingesta. 3. The cultivation of the habit of regular defæcation. 4. Plenty of exercise in the open air. 5. The administration of aperients, being at the same time careful to to avoid the *habit* of depending upon them. In many cases *tonics* combined with *nux vomica*, *belladonna*, or small doses of *aloes* are preferable when once the bowels have been opened by an aperient. Cascara sagrada (liquid extract ♏ x.-xx., *ter die*) is a very efficient remedy in chronic constipation. 6. Purgative *mineral waters*, as Carlsbad, Pullna, Hunyadi Janos, &c. (well diluted), are often useful. They should be taken before breakfast. 7. *Enemata* of glycerine (ʒ j.-ij.), or of soap and water (to which castor oil, &c., may be added) are sometimes of great service.

III. Melæna.

Melæna, or the discharge of blood per rectum, as already pointed out, always follows hæmorrhage into the stomach. But it may also be due to intestinal hæmorrhage, arising from causes similar to those mentioned when describing hæmatemesis.

When the bleeding is slight, comes from the small intestine, and is discharged slowly, the blood becomes altered, and the fæces are uniformly blackened or "tarry." If from the same source, but copious, and quickly removed, the tarry change is not observed. When coming from the rectum or the anus it is bright and unaltered, and *not* uniformly mixed with the fæces.

By attention to these points, and to the symptoms and signs associated with them, the cause and origin may usually be determined.

The special *treatment* of melæna must be conducted on the same principles as that of hæmatemesis. Oil of turpentine is especially useful. Fissures, ulcers at the anus, or hæmorrhoids require surgical interference.

IV. Colic.

Colic or painful spasmodic contractions of the bowels may arise from the following causes:—1. Irritation from improper or undigested food, altered secretions, foreign bodies, or retained fæces. 2. Organic disease and intestinal obstruction. 3. Reflex irritation from uterine or ovarian affections, &c. 4. Lead-poisoning. 5. Exposure to cold.

Symptoms.—The most characteristic feature of colic consists of griping, twisting *pains*, which generally begin about the navel and spread thence over the belly, *frequently change their position* and are *relieved by pressure*. The patient is doubled up, or tosses about restlessly. The belly is generally hard, tense, and retracted, but sometimes it is distended. The pain comes on suddenly, and presents remissions or intermissions. The bowels are usually constipated. There is *no fever*. These symptoms may be associated with faintness, nausea or vomiting. The *duration* is very variable.

TREATMENT.—It is often well to begin with an enema, either simple, or combined with castor oil or turpentine. In mild cases a brisk purgative may be administered by the mouth. A very convenient form is a full dose of castor oil with laudanum. When the stomach contains irritating matter, it should be emptied by an emetic. To relieve pain and spasm, opium with diffusible stimulants, such as spirits of chloroform, or tincture of cardamoms may be given; or morphia may be subcutaneously injected. Hot fomentations or turpentine stupes applied to the abdomen are useful adjuvants. The general conditions on which the colic depends subsequently requires attention.

INTESTINAL INFLAMMATION.

INTESTINAL CATARRH—ENTERITIS.

INTESTINAL CATARRH may be caused by errors in the quantity or quality of the food, or by everything which irritates the bowel. It is especially common in hot summer weather *(summer diarrhœa)*; but there is reason to believe that these cases are due to irritation set up by some specific micro-organism, though it is not yet proved. Catarrh of the bowel may also be caused by mechanical congestion from hepatic or cardiac disease. Sometimes the whole length of the gut is involved, especially in young children; or, as is more usual in adults, the mischief may be confined to special parts, indicated by such terms as *duodenitis*, *ileitis*, *colitis*, *typhlitis* (cæcum), and *proctitis* (rectum).

The morbid changes resemble those met with in gastric catarrh. Under certain rare circumstances a membranous exudation may form on the mucous lining of the bowel, and be discharged in shreds or casts. Local enteritis of a severe type may result from mechanical injury such as strangulated hernia, and intussusception, and in these cases the walls of the gut may be affected in their whole thickness, and become dark-coloured, thickened, and infiltrated with serum or pus, or coated with lymph on their serous aspect, and if not relieved, may ultimately become gangrenous. (Typhlitis and proctitis will be separately considered).

The *symptoms* of acute intestinal catarrh vary according to the degree and the seat of the inflammation. In most cases, but especially when the colon is affected, there are remittent colicky *pains* in the bowels, together with some little *tenderness* on pressure, flatulent distension, and borborygmi. *Diarrhœa* is usually present, the stools vary much in frequency and appearance, very rarely containing blood unless there be ulceration. The general health is apt to suffer, as indicated by fever, anæmia, depression, loss of appetite, and some emaciation.

Duodenitis is distinguished by the jaundice which accompanies it, other causes of jaundice having been excluded, as also by its frequent association with gastric catarrh. Catarrh confined to the ileum (*ileitis*) does not necessarily cause diarrhœa. The presence in the stools of unaltered bile, with a considerable amount of undigested food (especially fat), points to this variety. *Colitis* is indicated by tenesmus, the presence of mucus, unaltered blood, or pus in the stools, and tenderness over certain parts in the course of the colon.

The severer cases of inflammatory diarrhœa in children (*cholera infantum*) are due to acute catarrh of the whole gastro-intestinal tract, and are characterised by fever, vomiting, diarrhœa, the latter being at first fæculent, then slimy from admixture with mucus, and lastly profuse and watery, and rapidly followed by collapse.

In the limited intense form of inflammation involving all the coats, the gut becomes paralysed and its peristaltic movements cease, so that its contents cannot pass onwards. The symptoms therefore are those of acute obstruction, and will be described hereafter.

In *chronic intestinal catarrh* the main symptoms are chronic diarrhœa with liquid offensive or lienteric stools, with general indications of stomach derangement and malnutrition.

DIAGNOSIS.—*Simple diarrhœa* does not admit of any precise separation from that due to mild catarrh. The presence, however, of slimy mucus in the stools is a sure indication of inflammation of the colon. The features of *typhoid* and *dysentery* are sufficiently distinctive. In *colic* there is no fever. In *peritonitis* the pain and tenderness are much more marked, the bowels are *constipated*, and the constitutional disturbance much greater.

TYPHLITIS AND PERITYPHLITIS.

Typhlitis denotes an inflammation of the cæcum or the appendix in association with localised peritonitis. It is most frequent in young adult males. Such cases fall into one of two groups, according as the inflammation is *simple* or *suppurative*.

Simple typhlitis is the more common, and is nearly always the result of impaction of fæces either in the cæcum itself, or in the appendix. The symptoms are very similar in either case, but are usually more severe when the appendix is at fault. The morbid condition is that of a local enteritis involving the whole thickness of bowel-coats, coupled with local adhesive peritonitis. Ulceration is common, and sometimes leads to perforation, followed by rapidly fatal general peritonitis.

The symptoms are usually preceded by obstinate constipation, and consist of pain and tenderness in the right iliac region, together with slight fever. An ill-defined extensive tumour-like swelling quickly develops, which is often doughy on palpation. The bowels are confined, and sometimes also there is vomiting. The symptoms usually subside in from three to seven days, but are apt to recur on slight provocation for some time afterwards. Sudden onset of severe pain extending over the abdomen, with signs of collapse, indicate perforation of the bowel.

Suppurative typhlitis is almost always the result of an impacted fæcal concretion or foreign body in the appendix. Severe local peritonitis is set up, the appendix is perforated, and encysted intra-peritoneal suppuration occurs. This abscess may subsequently open into the anterior abdominal walls, the connective tissue behind the colon (burrowing in various directions), or into the cæcum or colon.

There is seldom any association with previous constipation. The symptoms often begin suddenly, and are much more severe than in the simple form of typhlitis. The fever is high and there is marked vomiting. The local pain is not only severe, but it radiates into the thigh and adjacent parts. There may also be some irritability of the bladder and rectum. The swelling is not obvious at first, and develops slowly, ultimately yielding—it may be—fluctuation on palpation. The fate of the abscess thus formed varies; but, if left to itself, it generally opens externally, in which case a chronic discharging fistula is not an unlikely sequel. Sometimes the progress of typhlitis exhibits a relapsing course—namely, attacks separated by variable intervals of marked improvement.

The diagnosis of typhlitis is by no means always easy, and the suppurative form may be closely simulated by pelvic, psoas, and peri-nephric abscesses.

The treatment in simple typhlitis consists of rest in bed, warm fomentations to the abdomen, an enema to clear out the rectum, followed by an occasional saline aperient combined with a full dose of tinct. belladonnæ. In more severe cases it may be necessary to give opium in some form to allay irritability and to relieve pain. In suppurative cases, operative interference should not be delayed, for the details of which the reader is referred to surgical text-books.

Proctitis and Periproctitis.

Proctitis or inflammation of the rectum may result from causes analogous to those of typhlitis, but far more often it is but part and parcel of some more general affection. Periproctitis is generally secondary to inflammation of neighbouring parts other than the bowel. It also occurs in connection with tubercular disease.

The chief *symptoms* are *tumefaction* and *induration* of the rectum (ascertainable by digital examination) or of the surrounding perineal tissue; with local *pain* and *tenderness* greatly aggravated on defæcation. When a perineal abscess has formed, it generally communicates with the rectum just within the external sphincter ani. It may subside spontaneously, or terminate in fistula The tubercular form is eminently unfavourable.

ULCERATION OF INTESTINES.

The various ulcers met with in the intestines may be divided into two groups. I. *Non-specific*, including (1) superficial and follicular ulcers from inflammation; and (2) duodenal round ulcer analogous to simple gastric ulcer. II. *Specific*, including (1) tubercular; (2) typhoid; (3) dysenteric; (4) cancerous; and (5) syphilitic.

The following *symptoms* (taken together) indicate ulceration—abdominal pain and tenderness, obstinate diarrhœa, and the presence of blood, pus, or mucous masses in the stools. But ulceration frequently occurs without any characteristic symptoms, especially when it affects the small intestine only.

TREATMENT OF INTESTINAL INFLAMMATION.

The *diet* must be light and unirritating. In simple catarrh, bismuth, oxide of zinc, and the alkaline carbonates are of service. It is not always prudent to check diarrhœa by opium or astringents. In those forms where irritating matter still remains in the bowels, a preliminary dose of castor oil is useful; and again, where it is associated with portal congestion, the diarrhœa should be rather encouraged than repressed. On the other hand *dysenteric diarrhœa* with blood or mucus in the stools, and *cholera infantum* generally call for opiates. For this purpose laudanum or Dover's powder may be usefully combined with nitrate of bismuth or oxide of zinc; or the opiate may be given in the form of an enema. *Chronic diarrhœa* often requires astringents, but the regulation of the diet (especially in young children) is of much greater importance. When there is *constipation*, purgatives should be only employed with caution; and if ulceration be suspected, it is better to avoid them altogether, and to trust entirely to opium. As substitutes for opium when this cannot be tolerated, henbane or conium may be tried. There can be no objection to emptying the lower bowel by simple enemata, whenever it may be necessary.

INTESTINAL OBSTRUCTION.

The numerous causes of intestinal obstruction (exclusive of malformation) may be classified as follows:—1. Causes acting within the gut—Impacted fæces, gall-stones, or foreign bodies. 2. Causes acting in the walls of the gut:—(*a*) Contracted cicatrix following ulceration. (*b*) Contraction due to inflammatory thickening. (*c*) Cancer. 3. Causes acting on the gut from without:—(*a*) Internal and external herniæ. (*b*) Compression by bands of lymph, tumours, or diverticula. 4. Causes resulting in altered relations of portions of the gut:—(*a*) Intussusception. (*b*) Twists or displacements.

Morbid anatomy and pathology.—Above the point of obstruction the intestine is greatly distended, while below this point it is empty and contracted. In acute strangulation or intussusception, peritonitis occurs, which is usually localised and very liable to end in gangrene. In chronic cases the muscular coat above the obstruction is hypertrophied, coupled with chronic catarrh of the mucous membrane.

Intussusception calls for separate consideration. It consists in the slipping of one part of the bowel into that immediately below it. The intussusception is thus made up of three tubes, the outer and middle of which are in contact by their mucous surfaces, and connected with each other at their upper ends; while the inner and middle tubes (*intussusceptum*) are in contact by their serous surfaces and connected at their lower ends. A fold of mesentery is imprisoned between the inner and middle tubes which drags the *intussusceptum* to one side, so that the orifice of the latter is turned towards the outer tube.

Two varieties may be distinguished. 1. One variety is only met with *post-mortem*, and is usually multiple. It is associated with the death-struggle, and causes no symptoms during life. 2. The other is the so-called *inflammatory intussusception*. The most common form is that in which the ileo-cæcal orifice descends into the cæcum, and as it passes into the colon drags down more and more of the ileum to an extent varying from a few inches to several feet, so that it may even protrude at the anus. Usually the ileo-cæcal valve forms the lowest part of the *intussusceptum*, but in rare cases part of the small intestine slips through this opening.

The after effects are as follows:—(1) *Compression* at the neck of invagination by the outer tube; (2) intense mechanical *congestion* followed by œdema or extravasations which still further increase the obstruction; (3) violent local *inflammation*, sometimes followed by (4) *gangrene* of the *intussusceptum*. Under the latter circumstance the *intussusceptum* may become detached, and afterwards be expelled by the rectum. Such an occurrence is not necessarily followed by rupture of the gut, this being prevented by inflammatory adhesions; but in such cases there is considerable subsequent risk of stricture at the point of union. The events above described occur with variable rapidity and severity according to the completeness of the obstruction.

General symptoms of obstruction.—1. *Vomiting* which is apt to become fæcal. 2. Griping *pains* of variable severity, associated with tenderness. 3. More or less tympanitis. 4. Obstinate constipation. 5. Hiccup. 6. Inflammatory signs, with small thready pulse, and tendency to collapse.

Diagnosis of the seat of obstruction may be aided by the following considerations. 1. *Upper part of small intestine.*—Obstructive symptoms are marked and appear early; the course is rapid; tympanitis is often absent, and when present is limited to the upper part of the abdomen and always relieved by vomiting; and the vomit though bilious is never fæculent. 2. *Lower part of ileum.*—Obstructive symptoms appear early, and the course is rapid; the tympanitis is marked, especially in front, the flanks being comparatively flat; and the vomit soon becomes semi-fæculent. 3. *Lower colon.*—Generally (but by no means always) the course is less rapid, and obstructive symptoms appear later; the tympanitis is extreme especially in the flanks; and the vomit is always fæculent before the close. Further information may also be obtained by digital examination of the lower bowel, by bougies, or injections. It is often said that scanty secretion of urine indicates obstruction high up in the bowel, while when lower down it is copious. Recent observations, however, seem to show that these differences have relation rather to the acuteness of the condition operating through the nervous system, than to the locality of the lesion.

Diagnosis of the nature of the obstruction.—Obstruction from strangulation, intussusception, or twists usually sets in suddenly, while in the vast majority of other cases the onset is gradual. An examination of the usual seats of external herniæ and of the rectum, serves to eliminate obstructive lesions in those parts.

Impaction by fæcal masses is most frequent in elderly people, is generally preceded by habitual constipation, and may be accompanied by a soft sausage-shaped painless abdominal tumour. It is sometimes complicated, however, with twisting or displacement of the loaded gut, which falls over by its own weight, and in this way the signs of acute strangulation may supervene. Impacted gall-stones may sometimes be diagnosed when elderly persons suddenly exhibit signs of obstruction with previous history of hepatic colic.

Constrictions and *compressions* are gradual in their progress, and are often preceded by gradually increasing constipation with (when affecting the lower bowel) small cylindrical stools. Aid may be derived from the previous history.

Strangulations are acute in their onset, and rapidly lead to complete obstruction followed by inflammation, gangrene, or perforation. They are more common over thirty years of age.

Intussusception is most common in children. The diagnosis rests upon the sudden onset, with violent intermittent colic, bloody mucous stools with tenesmus, association with a sausage-shaped abdominal tumour, and (in some cases) the detection of the intussusceptum by digital examination of the rectum. Intussusception high up in the bowels usually defies diagnosis.

Treatment.—At first, when the diagnosis is still uncertain, an enema may be given to relieve the constipation, but purgatives should be avoided. If nourishment cannot be retained in any form when taken by the mouth, nutrient enemata should be employed. Opium in these cases is our sheet-anchor. It should be given boldly and frequently, but with especial caution in children. It may be advantageously combined with full doses of belladonna. The *thirst* may be allayed by sucking ice. Hot fomentations or poultices applied to the abdomen are useful to relieve *pain*.

In cases of intussusception or displacement, endeavours may be made to reduce the obstruction by full and prolonged injection of warm water or air per rectum, combined with gentle kneading of the abdomen—the patient being previously put under the influence of chloroform. Finally surgical interference may be necessary for the gradual dilatation of certain fibroid strictures of the rectum, or to make an artificial anus, or to open the abdomen and reduce the obstruction.

CANCER OF THE INTESTINES.

Cancer of the intestines is almost always primary, and is most common in the rectum or sigmoid flexure, seldom occurring, however, in those under forty years of age. Various types of cancer are met with, but most frequently it takes the form of fungous out-growths from the mucous surface, displaying abundant tubular collections of cylindrical epithelium on microscopic examination. Colloid cancer comes next in order of frequency. As a result of infiltration and contraction of the bowel, intestinal stricture is of common occurrence. Secondary deposits may occur in the liver and other parts.

Symptoms.—*Pain* very variable in character and intensity, tenderness, habitual constipation or irregular action of the bowels, and tympanitis, constitute the usual symptoms. In some cases an abdominal tumour may be observed on palpation, but it is more often altogether wanting. Sooner or later the cancerous cachexia is usually manifested. Cancerous ulceration is indicated by diarrhœa, bloody mucous stools, and tenesmus; while stricture of the intestine necessarily gives rise to symptoms of obstruction. The symptoms of rectal cancer are often so insignificant, that a digital examination of the rectum should always be made on the least complaint of rectal discomfort in elderly persons. The duration of the disease is variable, but it seldom exceeds a year and a half; when death occurs from exhaustion, or from the secondary local effects of hæmorrhage, or from obstruction.

The *treatment* is purely palliative and symptomatic. An artificial opening into the colon may prolong life in some cases.

INTESTINAL TUBERCULOSIS.

Intestinal tuberculosis is rarely met with, except in connection with pulmonary phthisis. Generally tubercular mischief appears later in the bowel than in the lungs, but sometimes (especially in children) the sequence is reversed. The solitary and agminated follicles of the bowel are first attacked; they become enlarged and afterwards caseate and break down, forming an ulcer with indurated floor and margins. The ulceration extends much more rapidly in a transverse than in a longitudinal direction, so that it sometimes forms a complete ring around the inside of the gut. Tubercles are found in the tissue forming the edge and floor of the ulcer, which in their turn degenerate and break down, and constitute the mode by which the ulceration extends. Inflammatory adhesions form between adjacent coils of intestine at the seat of ulceration, and sometimes a perforative communication is set up between them. The ulcers have little tendency to heal, though occasionally imperfect cicatrisation occurs, and causes some intestinal constriction. Local and sometimes general tubercular peritonitis occurs, and there is always enlargement of the mesenteric glands, with or without caseation.

More or less abdominal pain and tenderness, with obstinate diarrhœa, and bloody stools observed in a patient suffering from phthisis would at once suggest tubercular implication of the intestine. But in many cases the symptoms point to nothing beyond intestinal catarrh. The affection rarely occurs in those under two years of age.

The course of the affection is progressively fatal, and treatment can only be palliative.

INTESTINAL WORMS.

The worms which infest the intestinal tract may be divided into two main groups.—1. The *Tæniada* or *Cestoda* or *Tape-worms* including (*a*) *Tænia solium*, (*b*) *T. mediocanellata*, and (*c*) *Bothriocephalus latus*. 2. The *Nematoda* or *Round worms* including (*a*) *Ascaris lumbricoides* (round worm), (*b*) *Oxyuris vermicularis* (thread worm), and (*c*) *Trichina spiralis*.

1. Tæniada.—*General Remarks.*—All members of this group have two phases of existence. Segments of the fully developed worm containing ripe ova become detached, and on being discharged from the body are scattered about. Sooner or later they find their way into the intestine of some appropriate animal. The embryo contained within each ovum then bursts its shell, passes through the intestinal wall, and works its way into the tissues. On reaching a suitable spot it further develops and presents a *head* resembling that

of a perfect worm, a *neck* and a vesicular or bladder-like appendage (*cyst*) filled with clear fluid. In this stage it is called a *cysticercus*. It possesses no sexual organs, and is only capable of futher development by its removal into the human intestine. This takes place when the tissue containing the cysticercus is eaten as food. Under these circumstances it fixes itself to the mucous membrane, drops its vesicular appendage, and develops from its hinder end the series of segments which characterise the adult worm. Thus *two distinct stages* are needed to complete the cycle of existence of these worms.

(*a*) *Tænia solium* or the common tape worm is of an elongated, flat shape, from five to thirty feet in length, and dwells in the small intestine. Its head (*scolex*) is about the size of a pin's head and is furnished with a circle of hooks surrounded by four suckers, by which it attaches itself to the intestine. From the head backwards it is made up of a series of soft white segments (*proglottides*), at first very small (*the neck*) but gradually enlarging as they become more mature. The segments of the neck are much broader than long, and nearly homogeneous; but proceeding backwards they gradually become longer than they are broad ($\frac{1}{2}$ in. × $\frac{1}{4}$ in.); and about one foot from the head, male and female sexual organs are visible, which open into a common aperture, and contain ova. Usually only one worm is present in the intestine at the same time.

Its cysticercus (*cyst. cellulosus*) gives the well-known appearance of "measly" pork, from eating which when badly cooked, tape-worms exclusively arise. The head exactly resembles that of tænia solium, and is joined to a roundish vesicle about the size of a pea by a short wrinkled neck.

(*b*) *Tænia mediocanellata* very closely resembles the former species, but it is longer, its head is three times as large, and it is not provided with hooklets. The *cysticercus* of this worm especially affects the ox, and is apt to be introduced into the human body by eating badly-cooked beef. It is of very common occurrence.

(*c*) *Bothriocephalus latus* is peculiar to certain parts of Europe and is the largest of all tape-worms. The head is club-shaped, with a longitudinal slit by which it attaches itself, and has no suckers or hooks. Its *cysticercus* is not definitely known, but is supposed to inhabit fish.

Symptoms.—In many cases there are no symptoms whatever. In others, however, there are indications of more or less nervous and digestive derangement. Thus pains in the bowels, irregular appetite, thirst, headache, troubled sleep, vertigo, mental depression or irritability, and itching of the anus or nose, are common symptoms. More rarely, the nervous disturbance assumes a severe type. The appearance of the ripe segments in the stools is characteristic.

Treatment.—The best way to get rid of the worm is to administer ʒ ss. to ʒ j. of the extract of the male fern early in the morning on an empty stomach, which should be shortly followed by a full dose of castor oil. Milk is a convenient vehicle for the drug. No absolute cure is effected unless the *head* is expelled. The bark of the pomegranate root, kousso, or oil of turpentine, have also been recommended, but are inferior to male fern.

2. Nematoda.—*General remarks.*—The members of this group are solid and rounded in shape, and possess a distinct alimentary canal provided with mouth and anus. The sexes are always separate. Much has yet to be learnt respecting the development of these worms, but it is certain that many of them exhibit a double phase of existence like *tæniada*.

(*a*) *Ascaris lumbricoides* or common round worm closely resembles, in its form, the common earth worm. The female is nearly twice as large as the male, measuring ten to fourteen inches in length. It is cylindrical, pointed at each end, of a white colour. The ova, of which thousands are discharged daily, are oval, about $\frac{1}{400}$ inch in diameter, with a thick irregular shell. More than five or six worms are rarely present at the same time. Their special habitat is the small intestine, but they are peculiarly apt to wander in every conceivable direction.

Symptoms.—These may be altogether wanting, or when present may be in no way characteristic, consisting chiefly of slight fever, disturbed sleep, thirst, and depraved appetite, with itching at the nose and anus. Occasionally a mass of them may give rise to temporary intestinal obstruction; or they may be vomited. Ova may be readily detected by the microscope in the stools.

Treatment.—The best remedy is *santonin* (gr. j.-iij., for a child, or twice this quantity for an adult), which should be given the first thing every morning on an empty stomach. An occasional dose of castor oil during the treatment is to be recommended.

(*b*) *Oxyuris vermicularis* or common thread worm is small, white, and thread-like. The female is twice the length of the male and measures from $\frac{1}{3}$ to $\frac{1}{2}$ inch. They dwell in the cæcum and colon, but sometimes appear to be confined to the rectum. They are the most common of all worms, are chiefly met with in badly fed children and always exist in great numbers. They are apt to migrate through the anus and may find their way into the vagina, setting up leucorrhœa or eczema.

Symptoms.—The most important sign is a troublesome itching about the anus, worse at night, and sometimes almost intolerable. There may be also itching at the nose, irritability of the bladder, digestive disturbance, and indifferent general health. The worms and their ova are readily detected in the fæces.

Treatment.—Enemata of salt and water, quassia, perchloride of iron, or lime water, repeated daily for a short period, are of most service. I have usually found it advantageous to administer internally, at the same time, santonin powders, every or every other morning, and the compound decoction of aloes (ʒ j. to ℥ j.) twice or three times a day, if there be no diarrhœa.

(*c*) Trichina spiralis*—*Trichinosis.*—This næmatode worm is met with in the muscular tissue, coiled up within an oval cyst, the walls of which are thick laminated, and semi-calcareous, each cyst being visible to the naked eye, as a minute granule. They are found chiefly in the flesh of pigs, and it is from eating trichinous pork that man is affected. The cysts are dissolved by the gastric juice, the embryos are liberated, and speedily reaching maturity, give birth to a vast number of living embryos which perforate the intestinal wall, and migrate to the tissues where they become again encapsuled.

Symptoms.—These at first consist of gastro-intestinal disturbance of an acute form not unlike typhoid fever, with coated tongue, diarrhœa, pain in the bowels, and fever. In the course of a few days signs of general muscular inflammation, viz., pain, tenderness, swelling, and stiffness of the voluntary muscles supervene. Dropsy, beginning in the face, and particularly in the eyelids, soon develops, extends to the limbs, and may subsequently become general.

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* Though not strictly speaking an intestinal worm, it is convenient to describe *trichina spiralis* in this place.

The duration and severity of trichinosis varies greatly. In mild cases recovery may take place in the course of a month, in others after three or four months.

In other cases again, the mortality is high, and death occurs from peritonitis, pneumonia, or general debility at any time between one and six weeks.

If the introduction of the parasites into the body be discovered quite early, prompt administration of a brisk aperient is likely to expel them before they have entered the tissues. Otherwise, the indications for treatment are purely symptomatic, as no specific remedy is known.

Sub-Section V.

DISEASES OF THE PERITONEUM.

PERITONITIS.

Peritonitis is almost invariably of secondary origin. Most commonly it may be traced to (1) external injury; (2) perforation or rupture of organs covered by peritoneum; (3) septic infection; (4) propagation of inflammation from other organs, viz.:—ulceration of the stomach or bowel, strangulation and intussusception of the bowel, typhlitis, metritis, &c.; or (5) new growths in the peritoneum, viz., tubercle or cancer.

Morbid Anatomy.—*Acute* peritonitis may be either general or local. Like inflammation of other serous membranes, congestion is followed by exudation of lymph with or without fluid. The lymph is yellowish and forms a layer of variable thickness over the membrane, and leads to adhesion between its opposed surfaces, which becomes more coherent with age. The fluid is usually scanty, more or less turbid, and generally holds flakes of lymph in suspension. Sometimes it is actually purulent, or it contains blood. *Chronic* peritonitis may also be either general or local. It is characterised by thickening and opacity of the membrane, with or without the effusion of fluid, while agglutinations and adhesions are apt to form and give rise to subsequent trouble.

SYMPTOMS.—The onset of acute diffuse peritonitis varies according to its cause. That of the puerperal and idiopathic forms is usually abrupt and attended with rigors; in the perforative variety it is characterised by sudden collapse; while in other cases—*e.g.*, after paracentesis abdominis, it is often very insidious. Whatever its mode of onset, the following symptoms quickly supervene. Severe pain with extreme tenderness sets in, which begins generally at one part and rapidly extends over the whole of the abdomen. The patient consequently lies on his back, with his knees drawn up, dreading the least pressure or movement of any kind. The tongue is coated and clammy, or red and irritable; there is great thirst; the appetite is lost; nausea and vomiting are often distressing; and the bowels are constipated as a rule, but are sometimes relaxed, especially in peritonitis of septic origin. The expression is one of extreme anxiety, and the features are pinched and drawn. There is *almost* always distinct fever; the pulse is rapid, weak, and small; the respiration is hurried and shallow; the urine is scanty and the bladder is sometimes very irritable. As a rule there is but little nervous disturbance, and the mind remains clear. The abdomen is tumid, hot and tympanitic; its respiratory movements cease; and the signs of fluid effusion (ascites) may be present.

In favourable cases the above symptoms gradually subside at the end of a few days, and convalescence sets in. On the other hand, in fatal cases, signs of rapidly increasing collapse supervene, though the pain and tympanitis may subside and disappear; the skin becomes cold and clammy; the pulse becomes more frequent, weaker, and often irregular; and low nervous symptoms set in, though the mind may be clear to the end. Lastly, it may become chronic.

The symptoms of *acute partial peritonitis* are essentially the same as those of the diffuse form, but are less marked, are more localised, and consequently vary according to the seat of the primary mischief.

The diagnosis of acute peritonitis mainly turns on the history of the case, the fever, the extreme superficial abdominal tenderness, the tympanitis, and extreme constitutional disturbance.

Treatment.—As a result of the recent great advance in the methods of abdominal surgery, the surgical treatment of peritonitis has attracted much attention. Apart from those cases in which there is some obvious surgical indication, as in acute local peritonitis from strangulated hernia, for instance, it is now considered desirable to open the abdominal cavity in all cases of perforative and purulent peritonitis, wash out and drain, provided they can be dealt with promptly. The technical skill and experience, however, which are necessary, must to a certain extent restrict the general adoption of such measures.

More often we have to fall back on three principal therapeutic measures, viz., perfect rest, free administration of opium, and local applications to the abdomen. The patient should be in bed in the position most comfortable to him, and protected from the pressure of the clothes by means of a bed-cradle. Opium in full doses (gr. $\frac{1}{2}$-1 every hour) or preferably hypodermic injections of morphia (gr. $\frac{1}{4}$-$\frac{1}{2}$ every three or four hours) should be administered, until contraction of the pupils clearly manifests its physiological action. Locally, if the constitution be good, and the case is taken in hand early, it is a good plan to apply from ten to twenty leeches on the abdomen; but such depletive measures should only be practised with caution. When they can be borne, hot and moist fomentations, poultices, sinapisms, or stupes, may be employed; while others again prefer evaporating lotions, cold compresses, and ice-bags. The food should be light, fluid, and nutritious, taken only in small quantities at a time, and frequently repeated. On the slightest threatening of collapse, alcohol should be exhibited. Under no circumstances should active purgatives be given; but in the *early* stages of peritonitis, saline aperients are sometimes beneficial.

Chronic general peritonitis may occur as the sequel of the acute form, but otherwise it is rarely observed, except in connection with cancer or tubercle of the peritoneum. In a *partial* form it frequently accompanies chronic disease of the abdominal viscera. It is characterised by opacity, thickening, and adhesions of the membrane, either with or without some fluid effusion. The chief symptoms to which it gives rise are—abdominal discomfort, colicky pains, tenderness, irregular action of the bowels, together with emaciation, debility, and other signs of constitutional disturbance.

TUBERCLE AND CANCER OF THE PERITONEUM.

General tubercular peritonitis seldom occurs alone, and is usually associated with phthisis or tubercle elsewhere. The tubercles are sometimes of the typical miliary form, and sometimes occur as large yellowish-white nodules rapidly undergoing fatty changes. The peritoneum exhibits the signs of inflammation already described. The disease is most frequent in young children.

Tubercular peritonitis as a rule develops insidiously, and is characterised by irregular fever of the hectic type. Occasionally, however, the disorder assumes a decidedly acute form. The symptoms are those of ordinary peritonitis, associated with the general manifestations of tuberculosis. Abdominal pain and tenderness are generally, but not always present. The quantity of effusion is sometimes quite small, but more often it is abundant. The effused fluid is turbid, and sometimes blood-stained; it is also remarkably apt to be shut off in closed cavities formed by intestinal adhesions (*encysted ascites*). Its course is tedious, and, as a rule, it terminates fatally. The diagnosis mainly rests on the proved existence of tubercle elsewhere. In the acuter forms it closely resembles typhoid fever.

The treatment is that already recommended for peritonitis and tuberculosis.

Cancer of the peritoneum is usually secondary to cancer elsewhere, and is commonly of the scirrhous type; but encephaloid and colloid cancers are sometimes met with. The symptoms are those of general peritonitis with or without effusion; together with an abdominal tumour or tumours, and indications of the cancerous cachexia. Abdominal pain and tenderness are well marked, and the effusion is frequently hæmorrhagic. The treatment is purely palliative.

TABES MESENTERICA.

Tabes mesenterica is a term applied both to scrofulous enlargement, and to tubercular disease of the mesenteric glands. The scrofulous form is essentially a disease of young children, and is generally secondary to chronic intestinal catarrh. The remarks on scrofulosis in a previous chapter must be applied to the affection now under consideration. True tubercular disease of these glands is either secondary to the strumous condition, or to tubercular ulceration of the intestine.

The symptoms of tabes are so generally masked with those of intestinal catarrh, or ulceration, and by other associated changes, that, in the absence of an abdominal tumour formed by the enlarged glands, it is impossible or difficult to definitely recognise them. A special feature of the disorder consists in the rapid and marked malnutrition which arises from interference with the entry of chyle into the thoracic duct.

ASCITES.

Ascites strictly implies a *dropsical* effusion into the peritoneum, but the term is very generally employed to indicate fluid effusions of *any* kind into that cavity. The causes of ascites may be grouped in three classes. It may arise (1) as a part of general dropsy due to cardiac, pulmonary, or renal disease; (2) from portal obstruction commonly caused by hepatic cirrhosis, or by pressure on the trunk of the portal vein in the hilum of the liver; or (3) from diseases of the peritoneum as inflammation, cancer, or tubercle. The amount of fluid varies from a few ounces to several gallons; and is usually clear, slightly viscid, alkaline, of a yellow or greenish tinge, and contains a large quantity of albumen. Its specific gravity is about 1015. When resulting from inflammation the fluid is turbid and flaky; and its specific gravity is higher.

Symptoms.—If the fluid be fairly abundant, we find on *inspection* that the abdomen is enlarged, its shape varying according to the posture. In the recumbent position the flanks are symmetrically bulged, and the belly is broad and somewhat flattened. The skin is smooth, tense, frequently marked with whitish striæ, and covered with a network of distended superficial veins. The navel is flattened out, obliterated, or everted. On *palpation* a peculiar thrill (fluctuation) may be felt on placing the hand flat on the abdomen, while the opposite side is smartly tapped with the fingers of the other hand. To get this result it is necessary that both points should be immediately over the effused fluid. The *percussion-note* is dull wherever the fluid is in contact with the abdominal walls. Further, since the fluid sinks to the most dependent parts, and the intestines float on the surface, the distribution of the dull and resonant areas varies according to posture. Lying on the back, the flanks are dull and the neighbourhood of the umbilicus is resonant; lying on one side the uppermost flank becomes resonant, and so on.

Other symptoms arise indirectly. Thus, dyspnœa and palpitation occur from pressure upon and displacement of the diaphragm and heart respectively. Pressure on the vena cava is apt to cause anasarca of the lower limbs and albuminuria, while congestion of the radicles of the portal vein always leads to functional derangements of the stomach and bowels.

Much, however, depends upon the amount of effusion. When it is scanty, recognition is difficult or impossible; and when the amount of fluid and consequent abdominal distension are excessive, the shape of the abdomen is more globular, and the dulness on percussion is more or less general in *all* positions.

Diagnosis.—Ascites may be confounded with an ovarian cyst, pregnancy, a distended bladder, a hydatid cyst, or a renal cyst; but space will not allow the distinctions from each to be mentioned in detail. Suffice it to say that the most typical features of ascites are, the symmetrical distension of the abdomen, a flattened navel, dulness in both flanks with resonance in front, the shifting of the limits of dulness on alteration of posture, the shape (concave upwards) of the upper line of dulness in the erect position and the results of mensuration. In simple ascites the maximum circumference of the belly is *never below* the level of the umbilicus, and the distance between the pubes and umbilicus is *never greater* than that between the umbilicus and the xiphoid cartilage. Lastly, the characters of the fluid (already given) afford valuable information.

Having estabilshed the existence of ascites it yet remains to ascertain its cause. In the first place it is necessary to separate inflammatory from simple dropsical effusions. Pyrexia, pain and tenderness of the abdomen, sweating and vomiting, are commonly present in greater or less degree in the former, but absent in the latter. The nature of the fluid in each also differs as already shown. Non-inflammatory dropsical effusions may be either renal, cardiac or pulmonary, or hepatic. In the *first* the ascites is preceded by anasarca which usually begins in the face, fluid is present as a rule in other serous cavities, the countenance is pale and pasty, and there are signs of renal disease. In the *second*, ascites is preceded by anasarca, which begins at the ankles and extends upwards, the countenance is dusky or livid, and there are signs of pulmonary or cardiac disease. In the *third*, anasarca is absent or follows the ascites, and is confined to the lower limbs; and there are the signs of hepatic disease. Cancer and cirrhosis are practically the chief hepatic diseases which are attended with ascites, and as to the diagnosis between them the reader is referred to another page.

The *treatment* of ascites for the most part is that of the affection on which it depends, but special measures are often called for, which essentially consist in the employment of diuretics, diaphoretics, and hydragogue purges. In the selection of such remedies, due regard should be paid to the general condition of the patient; thus strong diuretics, copaiba resin for instance, are out of the question in renal disease. Paracentesis abdominis may be resorted to in extreme effusions, but it should be borne in mind that the operation is sometimes followed by peritonitis, and that it is often worse than useless in ascites dependent on cancer.

SUB-SECTION VI.

DISEASES OF THE LIVER.

CONGESTION AND INFLAMMATION OF THE LIVER.

HEPATIC CONGESTION.

ACTIVE congestion of the liver always accompanies the digestive act, but whenever food is taken in excess, or is of an irritating nature, especially when connected with alcoholic abuse, it is apt to exceed its normal limits. Congestion also occurs in connection with malarial and other fevers, injury, or new growths. *Mechanical* congestion may be caused by any obstructive disease of the lungs or heart. *Passive* congestion may arise from sluggish portal circulation, associated with constipation or debility from any cause. Severe or long-continued congestion is apt to lead to diffuse inflammation of the liver.

Anatomical characters.—The liver is uniformly enlarged, dark-coloured, and gorged with blood.

In cases of well-marked mechanical congestion the centre of each lobule is dark from engorgement of the intra-lobular vein, while the periphery is relatively pale. This gives the surface of the liver on section a peculiar variegated appearance resembling a section of nutmeg *(nutmeg-liver)*.

In long-standing cases the cells which are compressed by the dilated capillaries become atrophied; moreover the interstitial connective tissue may be increased, which, from subsequent shrinking, leads to a diminution in the size of the liver.

Symptoms.—The subjects of hepatic congestion are irritable, depressed, and complain of headache and general malaise. Painful tension is felt in the right side, which often radiates to the right shoulder. The tongue is foul, the appetite is lost, nausea and vomiting are of common occurrence, and the bowels are irregular. The urine is scanty, high-coloured, deposits urates abundantly, and may contain a little bile. If jaundice be marked, there is probably some catarrh of the bile-ducts. The liver is uniformly enlarged, and tender on pressure. In chronic cases, ascites and other results of portal obstruction may supervene.

Acute Hepatic Inflammation—Hepatitis.

The chief causes of hepatitis are exposure to great heat (especially when associated with malarial influences), irregular habits of life, and spirit drinking. Except in connection with pyæmia, suppurative inflammation rarely occurs but in tropical climates, and it is usually secondary to dysentery.

Anatomical characters.—The process begins with active congestion. This is followed by infiltration of the affected part with leucocytes, and a development of embryonic tissue. The hepatic cells become swollen and cloudy. If suppuration occurs, one or more patches of softening make their appearance, which afterwards break down into pus. Murchison distinguishes two forms of hepatic suppuration:—(1) *pyæmic abscesses*, which are numerous, of a small size, and arise in a similar way to pyæmic abscesses elsewhere; and (2) *tropical abscesses*, which usually occur singly, and are often of large size. An abscess of the latter kind may burst into the peritoneal cavity, causing fatal peritonitis; but more often, as it approaches the surface of the gland, adhesive inflammation is set up, which glues the organ to adjacent parts, and the pus is discharged either externally, or into the lung, pleura, or stomach.

Symptoms.—In non-suppurative hepatitis there is nothing characteristic, the symptoms resembling a severe case of active congestion. The formation of pus is signalised by *chills* and *hectic* fever. If the abscess be superficial, *bulging* may occur in the right hypochondrium or epigastrium which soon yields a sensation of *fluctuation*, with signs of inflammatory change in the integuments. The *liver* is not only enlarged, but *altered in shape*, as may be ascertained by percussion. In doubtful cases, aspiration with a capillary trocar may be employed to aid the diagnosis.

INFLAMMATION OF THE BILE-DUCTS.

Catarrhal inflammation of the bile-ducts is a very common affection and is generally due to an extension of a similar process from the duodenum. It may also result from the irritation set up by passing gall-stones, or from hepatic congestion.

Anatomical characters.—The mucous lining of the ducts becomes swollen, congested, and discharges a slimy or semi-purulent secretion. The calibre of the duct thus becomes diminished, causing obstruction to the onward flow of bile. The lower part of the common bile-duct is most affected as a rule; and perhaps the most common cause of obstruction is the impaction of a plug of mucus in the orifice of the duct. Complete retention of bile is apt to be followed by acute or chronic hepatitis.

The *symptoms* are those of gastro-intestinal catarrh to which jaundice is superadded (refer to page 250). The physical signs are those of hepatic congestion.

GENERAL TREATMENT OF HEPATIC INFLAMMATION.

In the treatment of hepatic congestion and inflammation rest must be enjoined; the diet should be bland and unirritating; and stimulants as a rule are harmful. In the first instance, a mercurial followed up by some saline aperient, such as the sulphates of magnesia or soda, tartrate of potash, Hunyadi Janos water, and the like, will often be found useful. Local pain and tenderness may be relieved by fomentations or poultices, or (in severe cases) by the application of ten to twenty leeches to the abdomen. When less acute, iodide of potassium, chloride of ammonium, taraxacum, and nitro-muriatic acid are recommended. When the bile-ducts are involved, medicines directed to the gastro-duodenal catarrh are of service, such as soda, potash, or bismuth, in combination with rhubarb, ginger, or vegetable bitters.

If suppuration occurs, and the abscess can be got at, removal of the pus is desirable—by incision (with antiseptic precautions) when superficial, or by the aspirator and capillary trocar when deep-seated.

CIRRHOSIS OF THE LIVER.

Cirrhosis or "gin-drinker's" liver may be described as a chronic interstitial hepatitis, characterised by an overgrowth of the interlobular connective tissue, which tends to undergo cicatricial contraction, and thus to constrict and partly destroy the proper tissue of the liver with many of its ducts and vessels. It is most common in males between the ages of thirty and fifty; and is almost invariably induced by habitual spirit-drinking.

Anatomical Appearances.—In the early stages of the disease the liver is enlarged from the development of a soft vascular connective tissue (rich in young cells) in the course of Glisson's capsule, in the intervals between the lobules, and in the periphery of the lobules themselves. At a more advanced stage this new tissue becomes fibrous and contracts, and thus the liver not only becomes *smaller*, but altered in form. Its surface is more or less covered with prominences separated by depressions, which are sometimes small and fairly uniform in size (*granular*), but more often they are large, unequal in size, and separated by deep cicatricial contractions. The consistence of the organ is firm and dense, and the surface on section is also granular, and of a dirty-yellow colour.

The hepatic cells undergo fatty degeneration, and the lobular vessels and ducts are compressed and obliterated.

Symptoms and Course.—In the early stages the condition is similar to that described as arising from hepatic congestion.

Sooner or later, however, a series of characteristic events occur, which are traceable to the impeded circulation of the blood through the liver, caused by the vascular compression and obliteration above mentioned. Thus, there is (*a*) diminution in the area of hepatic dulness, often more marked over the left than the right lobe. (*b*) The surface is irregular (*hob-nail*) on palpation—when this is possible. (*c*) Ascites is very common. (*d*) The spleen is enlarged in a half of all cases. (*e*) The superficial veins of the abdomen are enlarged. (*f*) Hæmorrhoids are not uncommon. (*g*) Hæmorrhage into the stomach or bowels may occur. (*h*) Catarrh of the stomach and bowels with digestive disturbance in some degree is a constant feature. (*i*) Pain is usually absent at this stage. (*j*) Though decided jaundice is rare, a peculiar yellowish tinge of the skin is very often observed. (*k*) The urine is scanty, high-coloured, and deposits urates abundantly. (*l*) Cerebral symptoms may supervene. (*m*) There is in all cases progressive emaciation and debility.

Diagnosis.—In some cases a difficulty in diagnosis may arise where the ascites is so considerable as to prevent physical examination of the liver. Under such circumstances it may be confounded with cancer or tubercle of the peritoneum, but is distinguishable by (*a*) the enlargement of the spleen; (*b*) the condition of the urine; (*c*) the absence of marked pain or tenderness; (*d*) the absence of flakes of lymph in the ascitic fluid; and (*e*) by the history of spirit-drinking.

Treatment.—In early stages all stimulants should be avoided; the diet should be bland and simple; regular exercise in the open air is desirable; and the secretions require attention. *Medicinally*, iodide of potassium, chloride of ammonium, mineral acids, and vegetable bitters are principally recommended. Murchison speaks highly of mercurial treatment—green iodide of mercury (gr. $\frac{1}{2}$-i. *ter die*) internally, with inunction of iodide of mercury ointment over the right hypochondrium.

A few cases recover under treatment if taken early, but more often the disease proves fatal either from asthenia, peritonitis from frequent tapping, or a peculiar form of toxæmic coma.

In advanced stages, similar general principles are indicated. Spontaneous diarrhœa should not be checked unless excessive. To reduce the ascites, salines, and hydragogue aperients, and diuretics, such as the acetate or bitartrate of potash, with spirits of nitrous ether, digitalis, and decoction of broom, are useful. A pill containing mercury, squill, and digitalis, is a formula of great value, and the resin of copaiba (gr. x.-xv. *ter die*) in almond emulsion, is an excellent diuretic *when there is no albuminuria*. Quinine or iron, combined with digitalis, or the external application of linimentum hydrargyri, or fomentations of fresh infusion of digitalis, (four times B.P. strength) may prove useful in some cases. Should these measures fail, recourse to tapping is necessary. Acute gastric disturbance must be treated as occasion requires.

Note on other forms of chronic atrophy of the liver.—1. As a result of long-standing hepatic congestion from disease of the heart or lungs, a new formation of fibrous tissue with atrophy of hepatic cells takes place *in the centre* of the lobules. As this new tissue contracts and the atrophy proceeds, the liver diminishes in size, and its consistence becomes firm and dense.

It differs from cirrhosis as follows:—There is a previous history with present signs of cardiac or pulmonary disease; the ascites is *preceded* by dyspnœa and œdema of the legs; and there is no history of spirit-drinking.

2. The tissue of the liver may become indurated, or its surface may be marked with deep cicatricial contractions as the result of chronic inflammation of Glisson's capsule *(peri-hepatitis)*. This is *most common* in the subjects of constitutional *syphilis*.

3. An indurated and atrophic condition of the liver is sometimes met with after prolonged or repeated attacks of malarial fevers; and is characterised by dark pigmentation of its substance, and by the absence of any granulation of the surface *(red atrophy)*.

All these diseased conditions resemble each other in destroying to a greater or less extent the minute branches of the portal vein within the liver; which thus accounts for the general similarity of their symptoms during life, as a result of impeded portal circulation.

DEGENERATIONS OF THE LIVER.

Fatty Liver.

This affection is strictly speaking a *fatty infiltration*, consisting of the free deposit of oil-globules within the hepatic cells (page 20). It is met with in two very opposite conditions, (1) in large eaters and drinkers (especially of spirits) who lead indolent lives; and (2) in diseases attended with rapid emaciation, especially in *phthisis*. The paradox lies only on the surface. In both, the blood is loaded with fatty matter, traceable in the first case to an excessive supply of materials capable of being converted into fat; and in the second case to the patient's own tissues. The additional fact of respiratory interference explains the greater frequence of the affection in phthisis than in other wasting diseases.

Anatomical Characters.—The organ is enlarged and heavy; the margins are thick and rounded, the surface is smooth, the consistence is soft and doughy; and the surface on section is pale and greasy. *Microscopic examination* shows that the cells are enlarged, and loaded with fat globules. The process *begins at the circumference of the lobules* and extends inwards.

Symptoms.—The constitutional symptoms are few and not characteristic. The physical signs are (*a*)—smooth uniform hepatic enlargement; (*b*) soft rounded lower edge; (*c*) *no* pain, *no* ascites, and *no* jaundice.

Albuminoid or Lardaceous Liver.

As the above terms imply, this affection consists in a lardaceous infiltration of the hepatic tissue. It is met with especially as a result of (*a*) long-standing suppuration; (*b*) constitutional syphilis; (*c*) phthisis; and (*d*) chronic diseases which impair the general nutrition, such as ague, cancer, &c.

Anatomical Characters.—The organ is enlarged and heavier than normal, the edges are round and thickened, the surface is smooth; the consistence is firm and resistant; the surface on section is dry, glistening, and exhibits no trace of lobular structure and the affected tissue responds to chemical tests for lardaceous change before described (page 18). *Microscopical examination* reveals the characteristic change in the minute arteries and capillaries of the hepatic lobules. The process begins in the *middle zone of the lobules* corresponding with the distribution of the hepatic artery.

Symptoms.—As a rule these are not characteristic, and cannot be separated from those of the disease on which the degeneration depends.

The *physical signs* consist of smooth, uniform, hepatic enlargement, with firm rounded resistant lower edge. There is *no* pain or tenderness; and when jaundice or ascites is present it is due to pressure on the bile duct and portal vein respectively by lymphatic glands in the hilum of the liver, which are enlarged from lardaceous deposit. The implication of other organs with a similar deposit, notably the spleen, kidneys, and bowels, is of considerable diagnostic importance.

Treatment.—This chiefly merges in the treatment of the diseases with which the condition is associated. In albuminoid liver, tincture of iodine, iodide of potassium, iodide of iron, and chloride of ammonium have been more especially recommended.

HYDATID TUMOUR OF THE LIVER.

This affection is due to the development in the liver of the *cysticercal embryo* of a tape-worm, known as *Tænia echinococcus*, which infests dogs and wolves. The relationship between a cysticercus and a fully developed tape-worm will be understood on reference to the description already given of intestinal worms. It will then be seen that, contrary to the usual rule, it is the asexual cysticercus in this case which inhabits man, while its corresponding *Tænia* is found elsewhere. The ova discharged by the dog in the usual way are introduced into the human body through the medium of water or food, and on arriving in the stomach are set free, migrate, and usually settle in the liver. Sheep are affected in a similar way, and it is through eating their flesh that dogs become the seat of this tape-worm.

A hydatid tumour consists of a primary sac or vesicle (mother-cyst) with thick soft transparent and laminated walls, the innermost layer of which is formed by a delicate cellular membrane. The cavity of the mother-cyst is filled with clear limpid fluid of low specific gravity, which contains a large amount of chloride of sodium, but *no* albumen. Floating in this fluid are numerous "daughter-cysts" also containing fluid, and within the larger there may be a third and sometimes even a fourth generation of cysts. These secondary cysts have a similar structure to that of the primary cyst. Further, on the inner surface of the sacs several little prominences may be seen, about $\frac{1}{60}$ inch in height. These consist of a small vesicular body which is surmounted by a head furnished with four suckers, and a row of curved hooks, and is attached at its base to the cyst-wall by a narrow pedicle. The whole tumour thus constituted is enclosed by a zone of connective tissue of irritative origin. Such tumours usually occur singly, and vary indefinitely in size.

Modes of Termination.—1. In small tumours the parasite is apt to die; whereupon the cyst with its contents degenerates and shrinks. 2. The cyst may burst in any direction. 3. It may inflame and suppurate.

Symptoms.—Hydatid tumour of the liver usually runs a latent course, unless by reason of its size it presses upon or otherwise interferes with the function of surrounding parts.

Physical Signs.—(*a*) Gradual enlargement of the liver, which is not uniform, but irregular, so that the natural form of the organ is altered. (*b*) Occasionally there is bulging of the ribs. (*c*) When the tumour can be felt it is elastic and fluctuating, and as a rule it is quite painless.

It must be remembered that these signs vary according to the size, position, and progress of the tumour. In doubtful cases, an exploratory puncture may be made with a small trocar.

Treatment.—Where practicable an attempt should be made to remove the fluid with a fine trocar and canula, which may be employed either with or without aspiration. The removal even of a portion of the fluid has been found sufficient to cause the death of the parasite. Medicinal treatment beyond that which may be directed to the relief of symptoms is of no service whatever.

CANCER OF THE LIVER.

Cancer of the liver is usually *secondary* to cancer elsewhere, but it is occasionally *primary*. It is most often met with in those of middle or advanced life.

Anatomical Characters.—It usually occurs in the form of isolated rounded nodules, which are scattered through the organ, and vary greatly in size. Those which are superficial are flattened and depressed in the centre, and generally give rise to more or less local peritonitis. The nodules grow at the periphery, and at their centres soon undergo degeneration. The unaffected parts of the liver are congested and often stained with bile.

More rarely hepatic cancer takes the form of a diffuse infiltration; when the irregularity of surface, so characteristic of the vast majority of cases, is wanting.

Symptoms.—(*a*) The liver is *enlarged irregularly* as may be ascertained both by percussion and palpation. (*b*) It is *hard* and *resistant* to the touch. (*c*) It is both painful, and tender on pressure. (*d*) Jaundice and ascites are present in about half of all cases, and when once developed rarely disappear. These conditions are usually the result of pressure on the bile-duct and portal vein by enlarged glands in the portal fissure. (*e*) The spleen is rarely enlarged. (*f*) Constitutional symptoms, digestive disturbance, irregular action of the bowels, aching pains in the limbs, and progressive anæmia and emaciation (*cancerous cachexia*) are always present more or less. The urine is scanty, high-coloured, and deposits urates in abundance.

The *duration* rarely exceeds one year, and the *termination* is always fatal.

The *treatment* is necessarily purely palliative, and symptomatic.

TUBERCULOSIS OF THE LIVER.

This affection is never primary, and is only met with as part of general tuberculosis, or as secondary to chronic tubercle elsewhere. It cannot be recognised during life.

JAUNDICE.

Jaundice properly speaking is only a *symptom*, yet it is one of sufficient importance to merit a separate description. Jaundice denotes a yellow discolouration of the skin and conjunctiva due to impregnation with bile-pigment. Various discolourations of the skin in some measure resemble that of jaundice, but they can always be distinguished by the absence of bile-pigment from the urine, and by the usually natural colour of the conjunctiva.

Under ordinary circumstances we know that bile is being constantly absorbed by the vessels of the liver, gall-bladder, and intestines, and yet jaundice does not occur. The conclusion necessarily follows that it must undergo metamorphosis in the blood as soon as it is taken up. It is clear then that the accumulation of bile in the blood on which jaundice depends, will arise (1) when more bile enters the blood than can undergo metamorphosis, or (2) when there is any interference with the process of metamorphosis itself. In this way we arrive at a basis for the classification of the causes of jaundice.

I. JAUNDICE DUE TO EXCESS OF BILE IN THE BLOOD may be caused by:—1. *Obstruction of the bile duct* (*obstructive jaundice*), by (*a*) foreign bodies within it as gall-stones, &c.; (*b*) alteration in its walls from catarrhal inflammation, from cicatricial contraction after ulceration, or from new growths; or (*c*) pressure on the bile-duct from without by enlarged glands in the portal fissure, aneurism, or other tumours. 2. *Excessive secretion of bile*, such as occurs in hepatic congestion. 3. *Excessive absorption of bile* from habitual constipation.

II. JAUNDICE DUE TO CAUSES INTERFERING WITH THE METAMORPHOSIS OF BILE, viz.—1. *Poisons in the blood:*—(*a*) poisons of specific fevers (yellow fever, ague, &c.); (*b*) animal poisons (snake-bites, pyæmia); (*c*) mineral poisons, (phosphorus, copper, &c.); (*d*) acute atrophy of the liver (?). 2. *Impaired or deranged innervation:*—(*a*) severe mental emotion, fright, &c.; (*b*) concussion of the brain. 3. *Deficient oxygenation of the blood*, such as is met with occasionally in acute pneumonia, &c.

SYMPTOMS.—It will suffice to enumerate those symptoms which occur irrespectively of the cause of the jaundice. 1. *External discolouration.* As a result of impregnation with bile pigment, the tissues soon become stained with a yellowish or olive-green colour. This is well shown in the conjunctiva and skin. 2. The *urine* is also altered in colour from the presence of bile, and will stain the linen. The colour varies from saffron-yellow to brownish-black according to the quantity of bile which it contains. The chemical tests for bile in the urine are as follows:—(*a*) For *bile pigment*:—A drop or two of strong nitric acid added to a small quantity of urine containing bile, on a piece of white porcelain, will give a play of colours, passing through green, blue, violet, and red, into a dirty yellow. The addition of a few drops of a solution of iodine to urine containing bile, yields a green colour, and constitutes a very handy test. (*b*) For *bile acids*:—To about two drachms of urine in a test-tube add a *very little* cane-sugar or syrup, and afterwards pour in some strong sulphuric acid so slowly that the two liquids shall not mix. A *deep purple* colour is produced where the acid and urine meet, if bile acids be present. The urine also sometimes deposits crystals of leucin and tyrosin, on being evaporated down to a syrupy consistence. 3. A *bitter taste* is sometimes complained of. 4. *Derangements of digestion.*—When bile is absent from the intestines there is usually flatulence, constipation, and the motions are of a pale drab or clay colour. The digestion of fats is also interfered with. 5. Troublesome *itching of the skin* is often observed, and *cutaneous eruptions* of various kinds are not infrequent. 6. The *temperature* in the absence of any concurrent cause of fever, is slightly *lower* than normal. 7. The pulse is also slower, and may fall to fifty, forty or even twenty *per* minute. 8. Impairment of *general nutrition* is manifested by debility, emaciation, anæmia, and a not infrequent tendency to hæmorrhages from the various mucous membranes. 9. *Nervous symptoms*. There is always some languor, depression, and incapacity for exertion ; and sometimes signs of more severe disturbance are met with, such as delirium, stupor, coma, convulsions, and muscular tremors, attributable to toxæmia.

Diagnosis.—In practice it is important first of all to ascertain whether the bile-duct is obstructed or not. Where there is obstruction, the stools contain no bile, the jaundice speedily becomes intense, and an enlargement of the gall bladder can be sometimes detected ; but when there is no obstruction, the stools contain bile, the jaundice is slight, and the gall bladder is not enlarged. To prevent mistakes, it should be remembered (*a*) that jaundice primarily dependent on obstruction may persist for some time after the obstruction has yielded ; (*b*) that the obstruction may only be partial, and yet sufficient to give rise to jaundice ; and (*c*) that the presence of bile-stained urine or blood in the stools may give the latter a dark colour though obstruction exist. Jaundice which comes on *suddenly* is probably due either to obstruction of the duct by a gall-stone, or (when the signs of obstruction are wanting) to nervous causes. Intense jaundice which has developed *very gradually* probably results from pressure on the bile duct from without. Intermittent attacks of jaundice point to gall-stones in elderly people ; and to catarrh of the bile-ducts in children. Paroxysmal pain *preceding* jaundice points to gall-stones—*following* jaundice, to cancer. Pain and tenderness over the right hypochondrium concurring with jaundice, probably indicate (*a*) congestion, (*b*) catarrh of bile ducts, or (*c*) cancer. Jaundice with great enlargement of the liver, if the latter be painful and tender, is probably due to cancer ; if quite painless to lardaceous liver. When jaundice co-exists with ascites, there is usually either cancer or cirrhosis.

Febrile obstructive jaundice is almost always due to catarrh of the bile ducts.

ACUTE ATROPHY OF THE LIVER—MALIGNANT JAUNDICE.

The etiology of this rare disease is very obscure. It is most common in pregnant females, and rarely occurs in childhood or after thirty years of age. Intense emotion, syphilis, typhus, and intemperance, have been mentioned as predisposing causes. It is characterised by rapid atrophy and disintegration of the secreting tissue of the liver.

Symptoms.—More or less gastro-intestinal disturbance may precede the actual disease for several days. In other cases, the illness begins suddenly with *jaundice* (rarely intense), *vomiting*, *delirium*, and *convulsions*, followed by coma. There is a striking liability to *hæmorrhage*, indicated by petechiæ on the skin, epistaxis, hæmatemesis, and melæna. There is no fever. The bowels are almost always confined, and the stools are pale at first, but afterwards apt to become dark-coloured from admixture with blood. The urine is bilious and contains little or no urea, uric acid, or chlorides; and these matters appear to be replaced by *leucin* and *tyrosin.* Pain and tenderness in the neighbourhood of the epigastrium is generally complained of. Physical examination reveals a *rapidly progressive diminution in the size of the liver.*

The course is rapid, and the termination fatal. When it arises during pregnancy, abortion or miscarriage usually follows.

Treatment is generally of little avail, and must be necessarily symptomatic. Smart purgation has been recommended.

GALL-STONES.

Gall-stones or biliary concretions are met with in the gall bladder, or far more rarely in the biliary passages elsewhere. They may exist singly, but are more often multiple. Their size varies from a mere grain to a complete mould of the gall bladder. At first they are rounded in form, but when many gall-stones develop simultaneously, their opposed surfaces become flattened against each other. Taken together, the tendency is to form a mould of the cavity which they occupy. Their surfaces are commonly smooth, but sometimes tubercular; and they vary in colour from a pale yellow to a deep brownish-black. When fresh, they sink in water. Their consistence varies also, but they are usually soft enough to be cut with a knife. They consist mainly of *cholesterine* impregnated with pigment and lime salts, which is deposited around a small central nucleus, generally formed of particles of mucus. The cortex frequently presents a laminated structure.

The *effects* of gall-stones are various. 1. They may more or less completely fill the gall bladder, and yet cause no disturbance. 2. They may excite inflammation of the gall bladder and local peritonitis, leading to adhesion with adjacent parts—generally the bowel or abdominal wall. At a later stage ulceration may occur, followed by perforation and the direct escape of a calculus into the bowel. Or again suppuration may take place, burrowing into the abdominal wall, or bursting into the intestine. 3. More frequently, one or more gall-stones are dislodged, and escape into the ducts, and either pass permanently into the duodenum, or become impacted and lead to obstruction of the flow of bile.

The *symptoms* necessarily vary according to the circumstances just enumerated. 1. Nothing may be detected beyond a tense smooth rounded tumour due to distension of the gall bladder. 2. When inflammation occurs, we may expect to find pain, tenderness, and some fulness of this part, together with more or less febrile disturbance. 3. The passage of a gall-stone along the duct is indicated by griping, tearing pain in the belly, which is often excruciating (*hepatic colic*), lasting from a few hours to a day or so, and often accompanied by faintness, shivering, prostration, a small thready pulse, and vomiting. The pain is further characterised by its sudden onset, its usual recurrence two or three hours after a meal, and the relief given by pressure. The attack often ends as abruptly as it began, by the slipping back of the stone into the bladder, or its escape into the duodenum.

If the passage be at all protracted the flow of the bile is sufficiently impeded to lead to jaundice, which supervenes about twenty-four to thirty-six hours *after the pain*, and is accompanied by the usual signs of obstruction of the bile-ducts. Such attacks are apt to recur, but are usually shorter and milder than the first. The gall-stones may be found in the fæces by diluting the latter with water, and afterwards straining through a sieve.

Permanent impaction leads to enlargement and afterwards to atrophy of the liver, progressive impairment of nutrition, irregular abdominal pain, and ultimately to death from asthenia or some complication.

TREATMENT.—During the passage of a gall-stone, the pain is best relieved by hypodermic injection of morphia combined with a full dose of atropia (♏j.-iij.) repeated as necessary. In very severe cases it may be necessary to induce anæsthesia by chloroform. Hot fomentations should be applied to the abdomen, or a hot bath may be given.

Between the attacks much benefit may sometimes be derived from drachm doses of sulphate of soda, three times a day, well diluted. The success with which gall-stones have been removed by abdominal section justifies the consideration of such an operation in cases of severe recurrent hepatic colic.

SUB-SECTION VII.

DISEASES OF THE SPLEEN.

SPLENIC ENLARGEMENTS.

THE spleen is liable to many diseases; but since none of them can be recognised with certainty in the absence of enlargement, and since a splenic tumour is often the first and only indication of disease, it will be convenient to group them all under this head.

Enlargements of the spleen may either be acute or chronic. Acute enlargement may be due to inflammation or active congestion; while chronic enlargement may occur from mechanical congestion, simple hypertrophy, lardaceous degeneration, cancer, or hydatids. (Tubercle of the spleen also occurs either as a part of general miliary tuberculosis, or as secondary to chronic phthisis, but it cannot be recognised clinically).

The spleen normally lies in the left hypochondrium in the hollow of the diaphragm, and is partly over-lapped by lung, and partly in direct contact with the thoracic wall. It extends in the axillary line from the seventh rib above to the eleventh or twelfth rib below; and its anterior margin coincides roughly with the line of the ninth rib (fore part). It cannot be reached by palpation, and is only recognisable by percussion, the dull area varying much according to the amount of distension of the stomach and the bowels. For such reasons, slight degrees of splenic enlargement cannot be recognised with precision. The enlarged organ mainly extends downwards and inwards towards the pelvis and umbilicus, and may form a tumour of immense size, with protrusion of the abdominal walls. When once it has extended beyond the lower costal border it can be reached by palpation. We find that it follows the respiratory motion of the diaphragm, that it is movable, and that it shifts its position according to the posture of the body. Its anterior edge is well marked and presents one or more characteristic notches.

Inflammation of the spleen is exceedingly rare except as a result of embolism or pyæmia. Splenic embolism is generally associated with valvular disease of the heart, and forms one or more wedge-shaped hæmorrhagic masses in the substance of the organ, which are at first dark-red, but afterwards get paler, and finally almost white and cicatricial in character. More rarely the infarct becomes cheesy, or even suppurates. The peritoneum is usually inflamed over the affected part. Pyæmic infarcts are analogous to the foregoing, but are smaller and more numerous, and suppuration is the rule. The spleen as a whole presents a deep purple colour, and its tissue is very soft.

Pain and tenderness in the left hypochondrium, sympathetic vomiting, together with more or less splenic enlargement, occurring in the course of heart disease or pyæmia, would suggest the occurrence of splenic embolism.

Congestion of the spleen may be either active or mechanical. The former, as a morbid condition, chiefly occurs in connection with acute febrile disorders, especially malarial and typhoid fevers. The latter is a common result of obstructed circulation from hepatic, pulmonary, or cardiac disease. In congestion the organ is engorged with blood, and more or less enlarged, sometimes very much so. In continued or oft-repeated congestion chronic hypertrophy of the organ takes place.

The only indication of splenic congestion during life is enlargement of the organ, together with a sense of weight or tension, and some tenderness in the left hypochondrium.

Simple hypertrophy of the spleen is for the most part the result of chronic congestion. It is of common occurrence in the subjects of malarial affection, hepatic cirrhosis, and rickets. Frequently, however, no adequate cause can be found.

The spleen is enlarged from an increase in all the tissue-elements of the organ. This form of enlargement cannot be anatomically distinguished from that met with in leucocythæmia, which is described elsewhere.

Apart from leucocythæmia the symptoms of splenic hypertrophy are vague and unsatisfactory, and consist of a painless enlargement of the organ, anæmia, general debility, and a proneness to hæmorrhage from some one or other of the mucous surfaces.

Lardaceous degeneration of the spleen is of common occurrence, and is generally associated with a similar change in the liver, and other parts. As to its etiology and morbid characters, the general description already given may be referred to at page 18. It first affects the lymphoid trabeculæ of the Malpighian bodies. The affected organ is much enlarged, smooth, firm and heavy. On section the surface in the earlier stages, is studded with translucent granulations like boiled sago-grains (sago-spleen); but later on the degeneration is more or less uniformly distributed.

The recognition of this form of splenic enlargement, and its distinction from simple hypertrophy, can readily be made, as a rule, by the history of the case, and the evidence of a similar change in other organs.

Cancer of the spleen is very rare, and is almost always secondary to cancer of the stomach. It occurs in the form of nodular masses of the encephaloid type. The condition may readily be overlooked, owing to the indefinite character of the signs and symptoms to which it gives rise. Sometimes, however, it occasions a painful, tender, and irregularly-shaped tumour in the left hypochondrium.

Hydatids are exceptionally met with in the spleen, but the condition is not one which calls for any special description.

General treatment of splenic enlargements.—What little can be done concerns the treatment of the conditions to which the enlargement is due. Quinine and arsenic have often a marked influence in reducing enlargement traceable to malarial influence; but for the most part splenic enlargements are not amenable to treatment. In hypertrophy and lardaceous degeneration, iron, mineral acids, good food, change of air, and bathing followed by frictions, are indicated.

Section VII.

DISEASES OF THE URINARY SYSTEM.

THE URINE.

Renal disease and modifications in the characters of the urinary secretion are so intimately connected with each other, that a short account of the urine in health and disease is absolutely necessary.

Urine in health.—Healthy urine is a clear watery amber-coloured fluid, of acid reaction, with a specific gravity of about 1020. The average amount passed in the twenty-four hours is about fifty ounces in an adult, but it varies within wide limits according to the amount of fluid drunk, and the amount of water which escapes by other excretory channels. This amount contains about an ounce of urea, about ten grains of uric acid, and still less hippuric acid, creatin and creatinine. In addition it contains a considerable amount of chlorides, phosphates, and sulphates, of which the bases are soda, potash, lime, and magnesia, together with a minute quantity of oxalic and lactic acids.

On standing it undergoes certain fermentative and putrefactive changes. At first it becomes more acid, a result which is due to lactic acid fermentation; but after a variable period it becomes alkaline from the conversion of urea into carbonate of ammonia, is cloudy, and emits an offensive and characteristic odour.

Urine in disease.—*Physical characters.*—The urine is always scanty in febrile conditions, acute renal inflammation, or renal congestion; and in collapse it is sometimes almost or quite suppressed. On the other hand, it is abundant in diabetes, some forms of chronic renal disease, and after hysterical paroxysms. The specific gravity as a rule is in inverse relation to the quantity of urine, but sometimes we find a high specific gravity with abundant urine as in diabetes. The reaction is nearly always acid except in certain forms of dyspepsia, or where alkalies are taken in large quantity, or in vesical inflammation attended with premature decomposition of the urine. If the urine be turbid when passed, it is due either to the presence of earthy phosphates, mucus, or pus. In the first case the turbidity clears up at once on the addition of an acid. Pus may be distinguished from mucus by the presence of albumen in the former.

Urea is derived from destructive metamorphosis of the nitrogenous elements of the food and tissues. It is increased when food is taken in excess, in fevers, and in diabetes; it is diminished on the other hand, in all forms of marked chronic debility, in diseases of the liver attended with destruction of hepatic substance, and in most organic diseases of the kidney. A sudden diminution of urea occurring in the course of Bright's disease usually portends an attack of uræmia. The quantity is best ascertained by an apparatus (of which there are several cheap and efficient forms) based on the principle of the decomposition of the urea by hypobromite of soda, and measurement of the nitrogen evolved.

Uric acid and urates only occur as deposits in acid urines. Uric acid represents a certain stage in nitrogenous metamorphosis; when in excess, as in gout, it is often found as a reddish-brown, sandy, crystalline deposit. The crystals are generally lozenge-shaped, occurring singly or in clusters. It is important because it is apt to lead to the formation of urinary concretions. Urates of ammonia and soda often occur as an amorphous deposit in scanty acid urines, and though not necessarily morbid they generally indicate some hepatic, catarrhal, or febrile derangement. They are readily dissolved by heat or the addition of an alkali.

Leucin, *tyrosin*, and *cystin*, are exceptionally observed in the urine of disease, but the reader is referred to special text-books on the subject for their characters and the conditions under which they are found.

Salts.—The most important salts from a clinical point of view are the phosphates and oxalate of lime. *Phosphates.* Three varieties are found in the urine:—(1) Amorphous phosphate of lime, as a deposit, only occurs in alkaline or neutral urine. It does not dissolve on boiling, but a drop or so of nitric acid at once clears up the urine. (2) Ammonio-magnesian or triple phosphates are only thrown down in alkaline ammoniacal urine. They usually occur in the crystalline form of three-sided prisms with bevelled ends. (3) Stellar phosphate of lime is of rare occurrence, and assumes the form of crystalline rods or needles, single or grouped in rosettes. *Oxalate of lime* in small quantity is of no morbid significance, but when of habitual occurrence there is usually some obvious departure from health. Its crystals are octahedral or dumb-bell shaped. They are rarely found in other than acid urines.

Albumen in the urine in any quantity is always of pathological significance. It is found under one of three conditions; (*a*) structural diseases of the kidney; (*b*) renal congestion; and (*c*) the presence of blood or pus in the urine. The tests for albumen are based on the fact of its coagulation by heat or nitric acid. If urine containing albumen be boiled it becomes turbid, or a dense precipitate is thrown down (according to the amount present), which is *not* redissolved on the addition of a few drops of acetic or dilute nitric acid. Again, if a little urine containing albumen be poured gently down the side of a test-tube containing strong nitric acid, a white layer of coagulated albumen forms at the line of contact between the two fluids. A saturated solution of picric acid is also a most satisfactory re-agent; on addition to albuminous urine it yields a precipitate not redissolved by heat. The relative quantity of albumen in any specimen of urine may be fairly estimated by thoroughly boiling a small quantity of acidified urine, and then allowing it to stand for some time for the precipitate to subside. In forming an opinion as to whether albumen is the result of organic renal disease we should be mainly guided by the amount and duration of the albuminuria, and the associated presence of renal epithelium and casts.

Albumen is not infrequently found in the urine of those who appear to enjoy excellent health, and in whom there is no other evidence of renal disease. The somewhat questionable term of "*physiological albuminuria*" is applied to such cases. After making due allowance for certain cases in which such albuminuria may be the result of long antecedent organic renal disease thought to have been cured, and where it forms the initial symptom of organic renal disease as yet attended with no other symptom or sign, there still remains a certain proportion in which it is clearly due to functional causes. Of these the most important are derangements of digestion, nervous debility, and certain altered conditions of blood.

While we may admit the existence of albuminuria independent of renal disease, it seems certain that some of these cases ultimately lead to renal disease; and until it is possible to discriminate functional from organic cases more surely than at present, insurance societies will probably continue to reject *all* cases of albuminuria. The main features of the functional variety are—small quantity, intermittent appearance, absence of tube casts, normal heart, and good general health.

Grape-sugar in minute quantity is said to be often present even in normal urine, but its persistent presence in considerable amount is only found in diabetes. The general characters of diabetic urine will be described with that disease. The principal tests for grape-sugar are:—(1) *Fermentation test.* Urine to which a little yeast has been added, and allowed to stand for some time in a warm place ferments, if sugar be present, with the disengagement of carbonic acid gas. (2) *Moore's test* consists in boiling a small quantity of saccharine urine with an equal quantity of liquor potassæ, when a deep reddish-brown colour is developed. (3) *Fehling's test* consists in boiling a little of the suspected urine with rather more than an equal bulk of Fehling's solution (potassio-tartrate of copper) when a brick-dust precipitate of sub-oxide of copper is thrown down if sugar be present. The most convenient method of estimating the quantity of sugar passed in the twenty-four hours is as follows:—A sample of the collected urine should be fermented with yeast, and the specific gravity *before* fermentation should be compared with that *after* fermentation, and the difference in the number of degrees represents approximately the number of grains of sugar per ounce. A standard Fehling's solution may also be employed for the same purpose, the calculation being based on the quantity of urine necessary to decolourize a given measure of the copper solution.

Blood.—Blood in the urine (*hæmaturia*) may arise from hæmorrhage in any part of the urinary tract—(1) the kidney or its ducts; (2) the bladder; or (3) the urethra. In *renal hæmorrhage* the blood is uniformly diffused throughout the urine, which is of a dark smoky tint, and deposits a chocolate-coloured sediment on standing, in which blood-casts may often be found. Its causes may be grouped in three classes:—(*a*) *local lesions*—of which the more important are acute Bright's disease, cancer, calculous concretions, and acute congestion; (*b*) *symptomatic*—as in purpura, scurvy, and certain fevers; and more rarely (*c*) *supplementary*—as in sudden suppression of menstruation, or hæmorrhoidal flux. In hæmorrhage from the *bladder* and in rather free bleeding from the *ureter*, the blood is much less intimately mixed with the urine, in the former often appearing only towards the *end* of micturition, while the urine is of a pinkish or blood-red colour, and is associated with the symptoms of vesical disease. Hæmorrhage from the *urethra* is distinguished by its occurrence independently of micturition.

The detection of blood corpuscles by microscopic examination is the safest test for blood. The following is the best test for blood-pigment. Add a drop of tinct. guaiaci to a small quantity of urine in a test-tube, and shake it up with a little ozonic ether. If hæmoglobin be present, the ether, which after a few moments collects on the surface, exhibits a blue colour. The appearance of the blood-cells varies according to the specific gravity of the urine; if it be high the cells are small and shrivelled, if it be low they are large, transparent, and circular, in other cases the appearance may be normal. The urine is necessarily albuminous.

The *treatment* of hæmaturia will depend on its causation. If the hæmorrhage becomes serious in itself, cold applications to the loin or abdomen, the internal administration of turpentine, ergot, acetate of lead with opium, gallic acid, or sulphuric acid, and absolute rest are required.

Cases of bloody urine also occur, in which no corpuscles (or only very few) are to be seen, but the colouration is due to blood-pigment which has been separated out. This condition is termed Hæmatinuria or Hæmaglobinuria. It will be referred to again further on.

Mucus.—A light cloud of mucus is generally present in healthy urine, but the quantity is sometimes largely increased, especially in inflammation of the bladder. The urine is then very apt to become ammoniacal, turbid, and ropy. Between deposits of mucus and pus the gradation is imperceptible.

Pus.—Urine containing pus is turbid and does not clear on boiling, and can be readily recognised on microscopical examination. When present in quantity pus forms a yellowish white deposit, which becomes ropy on boiling with liquor potassæ. Pus in the urine may be derived from inflammation of any part of the urinary mucous tract, from renal abscess, or from direct communication with some neighbouring abscess.

Casts.—One of the most frequent accompaniments of inflammatory change in the kidneys is the formation of plastic moulds of the urinary tubules, fragments of which are washed onwards by the urine, where they may be detected on microscopic examination. They are of five kinds (1) *blood casts*, formed by coagulation of extravasated blood; (2) *epithelial casts*, formed by the agglomeration of desquamated epithelial cells or leucocytes; (3) *hyaline casts*, formed either by coagulated exudation of plasma, or albuminoid matter derived from renal epithelium; (4) *granular casts*, formed by fatty degeneration of hyaline casts; and (5) *fatty casts*, formed by fatty degeneration of epithelial casts.

URÆMIA.

The term uræmia is applied to an assemblage of symptoms which arises from an interference with urinary excretion, and is associated for the most part with organic renal disease, though not infrequently it is related to some obstruction in the outflow of urine. It probably depends on the retention within the system of the products of retrograde metamorphosis of the tissues and food.

The usual indications of uræmia consist of a peculiar ammoniacal odour of the breath, headache, drowsiness, muscular twitchings, dimness of sight, nausea or vomiting, and diarrhœa, succeeded by general convulsions or coma. The onset is generally insidious and marked by headache and drowsiness; but sometimes the patient is suddenly struck down with epileptoid convulsions, or insensibility, or he may become suddenly blind. Such cases are occasionally mistaken for apoplexy, epilepsy, or opium poisoning; hence the great importance of testing the urine carefully under these circumstances.

In treating uræmia every endeavour should be made to renew the flow of urine, and to excite the action of the skin. For the former—dry-cupping, with hot and moist applications over the loins; for the latter, warm baths, wet packs and subcutaneous injections of pilocarpine (gr. $\frac{1}{10}$ to $\frac{1}{20}$) are of great service. Free purgation with hydragogue cathartics, such as elaterium and compound jalap powder, is also indicated. When convulsions suddenly supervene, the inhalation of chloroform is useful; and in cases of sudden insensibility, venæsection may be employed, unless there is advanced chronic disease of the kidneys. Any removable cause of retention of urine of course requires special attention.

RENAL CONGESTION.

Congestion of the kidney may be either active or mechanical.

Active congestion merges into inflammation, of which it forms the early stage—by imperceptible gradations. It is a common condition in acute specific fevers, and also results from certain substances, such as turpentine or cantharides, when taken in excess. The kidneys are slightly enlarged, the bloodvessels are overful, and the renal epithelium may exhibit cloudy swelling. The urine is albuminous, is often accompanied with hyaline tube-casts, and sometimes by blood. There are no symptoms of importance.

Mechanical congestion generally depends upon venous obstruction from pulmonary or valvular disease. The kidneys are notably enlarged and engorged with blood; and, if the congestion is severe or long-continued, disintegration and degeneration of the renal epithelium follow, with over-growth of the interstitial connective tissue. The urine is scanty and high-coloured. A moderate amount of albumen is usually present. If the amount be large, or if associated with renal epithelium and casts, the kidneys must be assumed to have undergone some structural change.

The general principles to be observed in the *treatment* are absolute rest, bland unirritating food, warm and moist application to the loin, warm baths, and free purgation.

ACUTE BRIGHT'S DISEASE.

Acute Bright's disease is an acute inflammation of the kidney (*acute nephritis*). It commonly occurs as a sequela of scarlet fever, diphtheria, or some other acute specific fever; from exposure to cold; or in connection with pregnancy.

Morbid Anatomy.—The kidneys are more or less enlarged, and engorged with blood; but the surfaces are smooth, and the capsules remain thin and non-adherent. On section the colour is at first of a deep dusky-red throughout, the Malpighian bodies appearing as still darker points. At a later stage the cortex becomes paler or mottled, and contrasts strongly with the dark-red bases of the pyramids.

Microscopical examination reveals engorgement of the glomeruli with nuclear proliferation of the glomerular epithelium; the capsular space is more or less filled with fibrinous coagula enclosing leucocytes, red blood-cells, and granules; the convoluted tubules are choked with epithelium in various stages of disintegration, leucocytes, blood, and tube-casts; while in severe cases, there is inflammatory infiltration of the interstitial tissue of the cortex.

The *symptoms* essentially consist of fever, dropsy, and albuminuria. The affection usually begins with some chilliness or shivering, followed by general malaise, headache and possibly nausea or vomiting. There is more or less febrile disturbance for the first few days, thirst, loss of appetite, constipation, and occasionally pain or tenderness in the loins. The *urine* is very scanty, turbid, abnormally dark or smoky in colour, with a high specific gravity, and contains albumen, blood, and a diminished quantity of urea. On standing it deposits a copious sediment, which on microscopic examination, is found to consist of blood-cells, loose renal epithelium, epithelial hyaline granular or blood-casts, and amorphous urates. *Dropsy* is not invariably present, but when it occurs it generally comes on early, and is sometimes the first indication of mischief. It begins in the eyelids or scrotum, and rapidly becomes more or less general. The serous cavities and internal organs are also liable to be involved in the dropsical process.

The progress and results of acute Bright's disease vary greatly. In favourable cases (which constitute the large majority) the symptoms subside in the course of a few weeks, the anasarca usually disappearing long before the albumen has quite vanished from the urine, and convalescence is gradually re-established. On the other hand, it may become chronic; or death may occur

(1) from inflammation of the lungs, pleura, or peritoneum; (2) from anæmia or asthenia; (3) from œdema of the lungs or larynx; or (4) from the supervention of uræmia. Owing to the tendency to recurrence, great care should be taken until the albuminuria has quite disappeared.

TREATMENT.—The patient should be kept warm in bed at an equable temperature. In the early stages the bowels should be kept fairly open: the action of the skin should be maintained by occasional warm water or vapour baths; and dry cupping or hot fomentations may be employed over the loins. Medicinally, full doses of the acetate or citrate of potash, well diluted, together with digitalis and nitric ether are of service. As the more acute symptoms subside, vegetable tonics and iron are indicated. The diet should be light, nutritious, and unstimulating, and alcohol is rarely needed. No restraint should be exercised with regard to the amount of water to be taken, for the more freely the better. If the dropsy be so excessive as urgently to call for relief, acupuncture may be resorted to. Only one or two punctures should be made, owing to the readiness with which erysipelatous inflammation takes place. Complications must be treated as they arise.

CHRONIC BRIGHT'S DISEASE.

CHRONIC Bright's disease occurs either as the sequel of an acute attack, or is chronic from the outset. In the latter case, it must be regarded as the expression of a general malnutrition, which is sometimes traceable to one cause, and sometimes to another. Irregular living, frequent exposure to cold and damp, intemperance, dyspepsia, gout, and plumbism are common remote causes of this affection. The essentially chronic forms are most common in the latter half of life.

PATHOLOGY AND MORBID ANATOMY.—The renal changes are essentially of a chronic inflammatory nature, and in all cases tend to become diffuse and to affect the organ as a whole. Clinical observation, however, justifies the recognition of two forms, according as the tubular or parenchymatous portion of the kidney, or its interstitial connective tissue, is first and mainly affected. To the former is given the name of the "smooth white kidney," to the latter that of "granular" or "contracted kidney."

The *smooth white kidney*, whether preceded by acute inflammation or not, presents appearances which do not essentially differ from those already described as pertaining to the later stages of acute Bright's disease. The weight of the organ may be doubled, its surface is smooth and pale, and the capsule is not adherent. On section the cortex is seen to be much thickened, pale or yellowish, and the pyramids too may be paler than normal. On microscopic examination, the tubes are found stuffed with epithelium in various stages of disintegration and often markedly fatty; while here and there they contain fibrinous material. In a certain proportion of cases, especially in those supervening on acute scarlatinal nephritis, the interstitial tissue is largely implicated, and if life be sufficiently prolonged, subsequent contraction and atrophy bring about a condition which is difficult to distinguish from true granular kidney.

The *granular* or *contracted kidney*, in its typical form, is small, its surface is granular, or nodulated, and its capsule is firmly adherent. On section the cortex is seen to be of a red colour, and much thinner than normal; the tissue generally is hard and resistant, and may be studded with small cysts. On microscopic examination, the connective tissue is relatively in great excess, and the tubules are variously altered—some are denuded of epithelium and distended with fibrinous material or greatly atrophied, while others are little changed. The primary change thus appears to consist of proliferation with subsequent contraction of the interstitial tissue, especially in the neighbourhood of the Malpighian bodies, the tubules with their epithelium being involved at a later period. In both forms of chronic Bright's disease, but especially in the "granular kidney," cardio-vascular changes are very common, characterised by hypertrophy of the heart, and thickening of the small arteries. Cardiac hypertrophy has no necessary casual relation with the arterial change, and not infrequently occurs independently of it. It is due to stimulation of the heart from morbid substances in the blood, resulting from defective elimination, and to increased capillary resistance probably caused by alteration in the density of the blood (Saundby). The arterial changes are the outcome of chronic arteritis induced by irritation from altered blood. The arteries, moreover, are prone to atheromatous degeneration.

General Symptomatology.—Excluding those cases of chronic Bright's disease which occur as the sequel of an acute attack, the onset is usually very insidious, and characterised by frequent micturition (especially at night), gradual failure of strength, increasing pallor, a little fulness about the ankles at night or puffiness of the eyelids in the morning. More rarely, the disease is suddenly revealed by the supervention of coma or general convulsions.

When fully established, the following constitute the more important symptoms:—Albuminous urine containing tube-casts; more or less œdema or dropsical effusion; pulse of high tension; anæmia and shortness of breath; dryness of the skin; digestive derangements;

headache and giddiness, together with a tendency to dimness of sight and retinitis, uræmia, secondary inflammations, and cardiac hypertrophy. The disease is essentially chronic, and is subject to sub-acute exacerbations from time to time. Death ultimately takes place from uræmia, œdema of the lungs or larynx, secondary inflammations, asthenia, or the occurrence of apoplexy or phthisis.

SMOOTH WHITE KIDNEY—CHRONIC TUBAL NEPHRITIS.—Except in those cases which supervene upon acute nephritis, the onset is insidious, and for the most part is characterised by progressive weakness, impaired nutrition, pallor, a pulse of high tension, puffiness of the eyelids and ankles, loss of appetite, and frequent micturition. As the case proceeds, the pallor increases, and the dropsy is abundant. The urine is scanty, its specific gravity is normal or slightly increased, it is turbid or smoky, highly albuminous, deficient urea, and on standing it deposits a copious sediment containing renal epithelium, leucocytes, sometimes blood, together with epithelial fatty granular or hyaline tube-casts. The termination is almost always fatal, with an average duration of under one year. As to complications, secondary internal inflammations are more common than uræmia. Such cases are most common in middle life.

GRANULAR KIDNEY is almost always chronic from the commencement and begins very insidiously. The *average* age at which it occurs is forty to fifty years. The urine is copious, of low specific gravity, clear, contains but little albumen, and on standing deposits a very slight sediment containing a few hyaline or granular casts, and some epithelium. Dropsy is altogether absent in many cases, and when present is usually slight, and limited to some puffiness of the eyelids or ankles. The face though pale, is not white or pasty. The course is much more chronic, there is less tendency to secondary inflammations, and uræmia is more frequent than in the former variety, while hypertrophy of the heart and thickened arteries are more commonly found. The latter are apt to become degenerated, and thus may lead to apoplexy. The prognosis is ultimately very unfavourable.

The TREATMENT of chronic Bright's disease is not satisfactory. The body should be kept warm and be clothed in flannel. All exposure to cold must be carefully avoided, and residence in a warm equable climate is desirable. The food should be light, bland, and nutritious; and a diet of milk or skimmed milk often answers better than anything else. Alcohol is not necessary as a rule, but sometimes digestion is carried on more efficiently with the aid of a little light wine, such as claret or hock. Medicinally, iron given persistently, and in full doses, with or without quinine, has perhaps the most beneficial effect. The tincture of the perchloride is the best preparation, but where this disagrees with digestion, it must give place to the blander ammonio-citrate or tartrate of iron. Extreme dropsy is most effectually combated by purgatives and diaphoretics. Of the former, compound jalap powder, bitartrate of potash, elaterium, and gamboge are among the best; while the action of the skin may be excited by warm baths or warm wet-sheet packs. Sometimes puncture or incision of dropsical parts gives great relief, but from the readiness with which erysipelatous inflammation takes place, it requires great caution in its employment. The treatment of uræmia has already been alluded to, and secondary inflammations must be dealt with as they arise.

SECONDARY NEPHRITIS.

Under this head are grouped those forms of nephritis variously described as surgical kidney, consecutive or ascending nephritis, and puerperal nephritis. Although secondary nephritis is—from a pathological stand-point—closely related to Bright's disease proper, yet on clinical grounds it seems desirable to consider it separately. It is the result of obstruction in some part of the urinary passages below the kidney, and is associated most often with pregnancy, stricture of the urethra, enlarged prostate, or tumours of the uterus, ovaries or bladder. Under the influence of chill or surgical operations such as catheterism, &c., the renal inflammation is liable to acute exacerbations. It rarely occurs before middle life, except in connection with pregnancy.

At first the kidneys are merely enlarged and congested. Afterwards they become paler in colour, tough and flabby. The capsule is generally adherent, and the surface of the organ may be granular. The renal substance, here and there, is apt to show signs of suppurative change. Still later, the kidney may become contracted. The morbid changes are often unequal on each side. More or less pyelitis is always present. The microscopic appearances are for the most part those of tubal and granular nephritis variously combined. Cardiac hypertrophy and arterial thickening may be observed.

The symptoms are to a large extent masked and modified according to the condition to which the nephritis is secondary, and the renal disease often escapes recognition until some acute exacerbation occurs. The character of the urine also varies according as the nephritis is chronic or acute, or associated with cystitis; but it always contains albumen and tube-casts. In the absence of acute nephritis, dropsy is usually absent or moderate except in pregnancy, when it is principally due to concurrent pressure on the large abdominal veins. Unless the primary cause is removed, the patient usually lapses into a typhoid state and dies. Sometimes uræmia occurs, especially in cases associated with pregnancy.

The *treatment* is very unsatisfactory, unless (as in pregnancy) the primary cause can be removed. It should be remembered that surgical interference of any kind is very badly borne in these cases.

METASTATIC ABSCESS OF THE KIDNEY.

Abscesses of this kind are due to pyæmic infection, and therefore are more or less masked by the general disturbance due to the primary disease. They are usually numerous and of small size; occurring more abundantly in the cortex of the organ, and generally affecting both kidneys. Their mode of origin was referred to in the chapter on pyæmia. Their existence may be suspected when in the course of pyæmia, we observe pain and tenderness in the loins, with scanty acid urine containing epithelial and granular casts.

PYELITIS AND PYO-NEPHROSIS.

Catarrhal inflammation of the pelvis of the kidney, or pyelitis, most frequently arises from the irritation set up by the presence of a renal calculus, or by retention of decomposed urine. Less frequently it is due to extension of inflammation from the bladder, or to the elimination of medicinal irritants (such as turpentine and cantharides), or to mere exposure to cold. Chronic pyelitis is also apt to occur in gouty and lithæmic subjects, from the persistent irritation set up by highly acid urine.

Morbid Anatomy.—The *acute* form is characterised by redness, swelling, and ecchymosis of the lining mucous membrane with shedding of its epithelium, and a discharge of muco-pus. In more *chronic* cases the membrane is gray or slate-coloured, much thickened, and sometimes ulcerated or covered with a layer of phosphatic deposit. The opening into the ureter, already much narrowed by inflammation, is very apt to become permanently blocked by the impaction within it of a calculus, a blood-clot, or thickened pus. Under these circumstances the pelvis becomes greatly dilated, and its cavity gradually encroaches on the substance of the kidney which atrophies, and is ultimately converted into a multilocular sac filled with pus and decomposing urine (*pyo-nephrosis*). The abscess thus formed may burst into the cellular tissue round the kidney, and set up a peri-nephric abscess, which burrows in various directions, or the whole mass may become inspissated and more or less calcified.

The *symptoms* immediately due to the pelvic inflammation are an aching pain or weakness in one or both loins, with tenderness on pressure. At first the urine is acid, and often contains a little blood or pus with pelvic epithelium (cells spindle-shaped, tailed, elongated, or irregularly rounded). Later on the urine sometimes becomes ammoniacal and contains pus in large quantity. When the ureter is blocked the urine may be temporarily quite normal; but should the obstacle give way, it becomes suddenly loaded with pus. Micturition is too frequent. Later on a renal tumour may be observed, which is variable in size, tender on pressure, and obscurely fluctuating. More or less febrile disturbance of the hectic type is commonly present, with diarrhœa, or constipation (due to pressure on the colon).

The *prognosis* of acute pyelitis is usually favourable unless it be very severe. The terminations of chronic pyelitis vary widely according to its cause, the constitutional condition, and the complications. Pyo-nephrosis is almost always fatal sooner or later if left alone.

The *diagnosis* of pyelitis as a complication of cystitis, enlarged prostate, or old urethral stricture, and in the absence of a renal tumour is often impossible. The presence of pus in the urine and irregularly-shaped epithelia, independently of vesical prostatic or urethral disease—especially when associated with tenderness or aching in either loin—would indicate pyelitis. When associated with vesical catarrh the diagnosis of pyelitis becomes very difficult. The presence of a tender, throbbing renal tumour, the sudden appearance or disappearance of pus in the urine, with fever and shivering, make the diagnosis comparatively simple.

The *treatment* for the most part consists in ascertaining and removing the cause when this is possible. In acute cases, rest in bed, diluent drinks and salines are indicated in the first instance. Poultices or fomentations to the loin may be employed to relieve the pain—or opium in severe cases. When the disease has become chronic, benzoic acid, terebinthinates, perchloride of iron, tannic acid or acetate of lead may prove useful. In gouty pyelitis, alkalies with vegetable bitters are of service, while in other cases nitro-muriatic acid answers better. Nutritious food and generally healthy bracing surroundings, are always indicated. Considerable success has recently attended the surgical treatment of these cases, and therefore when the diagnosis is assured, the abscess may be opened from the loin.

PERI-NEPHRIC ABSCESS.

We have already seen that suppuration, beginning in the pelvis of the kidney or in the renal substance, may extend beyond the limits of the organ into the surrounding tissue. In other cases it is due to extension upward of peri-typhlitis or pelvic cellulitis, or to traumatic injury. The onset is very insidious, and until a tumour has formed, the diagnosis is very difficult. It differs from renal suppuration mainly in the normal condition of the urine, and greater superficiality of the pain, tenderness and swelling. The abscess (if not interfered with) may burst externally in the loin or at Poupart's ligament; less often, it burrows in an upward direction towards the thorax; and quite rarely, it opens into the peritoneum.

The *treatment* consists of free incision or aspiration as soon as the abscess is sufficiently superficial.

HYDRO-NEPHROSIS.

A sudden and complete obstruction of the ureter sometimes leads to hydro-nephrosis. This condition is characterised by progressive dilatation of the duct above the obstruction, followed by compression and atrophy of the renal substance, with distension of its fibrous capsule, resulting in the formation of a sacculated chamber, sometimes of enormous size, which is filled with fluid. The latter consists of altered urine, is pale, watery, of low specific gravity, but not albuminous unless some pus or blood be present. The sac rarely bursts, but it either remains as a chronic cystic tumour, or the obstacle becomes dislodged, and is followed by discharge of the fluid, *per vias naturales*, and shrivelling up of the remains of the organ. As a rule only one kidney is affected, and the other becomes hypertrophied. The affection is sometimes congenital, as the result of an imperforate ureter.

The only sign of hydro-nephrosis is the formation of a painless, soft, fluctuating tumour occupying the loin, and displacing the colon which lies in front of it. A sudden subsidence of the tumour with a copious discharge of somewhat altered urine, though very rare, is highly characteristic.

If by reason of its size any *treatment* is required, it is purely of a surgical nature. Relief can be attained by aspiration, but a radical cure is possible by abdominal section and removal, an operation which has been done successfully within my own experience.

LARDACEOUS KIDNEY.

On pathological grounds lardaceous kidney should be distinguished from chronic Bright's disease, notwithstanding their clinical relationship. The conditions under which it occurs, and its morbid characters are identical with those of lardaceous degeneration elsewhere (page 18). The kidney is smooth, pale, and gradually increases in size and weight. On section, the cortex is thicker, and the Malpighian tufts in which the change first begins appear as glistening points. At a later stage the renal substance as a whole becomes more or less uniformly waxy. The liver and spleen are usually concurrently affected.

When the degeneration has sufficiently advanced, the urine is increased in quantity, pale, with a specific gravity from about 1012 to 1015, is more or less albuminous, and may contain a few hyaline or granular casts. Dropsy as a rule is not well-marked, but is commonly present. Micturition is frequent. It differs from the granular form of Bright's disease in the absence of cardio-vascular hypertrophy, the slight tendency to uræmic phenomena, and its common association with lardaceous changes in other organs—liver, spleen, and intestines; the specific gravity of the urine is also somewhat higher, and the excretion of urea is not so much reduced. In its later stages, owing to diffuse inflammatory changes set up in the kidney, the symptoms resemble those of ordinary chronic Bright's disease, and call for similar treatment.

CANCER OF THE KIDNEY.

Cancer of the kidney may be either primary or secondary. Primary cancer is almost always of the soft or encephaloid type, and occurs either in nodules or as a diffuse infiltration. As a rule only one kidney is affected, but secondary deposits are apt to occur in the neighbouring glands. It is most common in young children, but it also occurs less frequently in adults. Secondary cancer affects *both* kidneys, and rarely causes much enlargement, thus rendering it difficult to be diagnosed during life.

The main *symptoms* of primary renal cancer consist of a renal tumour, hæmaturia, and general constitutional disturbance. The tumour occupies one or other loin, grows rapidly, and generally attains a very large size, It is rounded but irregular in outline, and generally of firm but unequal consistence. It is almost invariably *crossed by the colon;* and owing to adhesion with surrounding parts it is immovable. In some cases it is painless, but as a rule there is severe lumbar pain and tenderness from the onset. The superficial abdominal veins are enlarged, and the lower limbs may become œdematous from pressure on the vena cava. Hæmaturia when present is a sign of very great value; but it is absent in about half of all cases—more often so in children. Hæmaturia occurring in elderly persons (whether a tumour is present or not), of intermittent duration, not specially related to bodily movement, and not attended with increased pains, should always excite a strong suspicion of cancer. The constitutional symptoms are those which generally accompany cancerous growths. Death occurs from exhaustion. The average duration in children is about seven months, and in adults about two and a half years.

The *treatment* is purely palliative and symptomatic.

TUBERCLE OF THE KIDNEY.

Tubercles may occur in the kidney as part of acute general tuberculosis; but under these circumstances they are generally few in number, the patient dies before further local changes take place, and diagnosis of the renal condition is not possible.

On the other hand, generally in association with tubercular mischief elsewhere, renal tuberculosis may run a more chronic course, and lead to destructive inflammation of the organ. The tubercles first appear as gray granulations in the renal substance, which afterwards coalesce into cheesy masses. The latter soften and break down, forming pseudo-abscess cavities, which gradually extend by ulceration. The kidney becomes moderately enlarged, and is ultimately converted into a fibroid sac, divided by septa, with little or no trace of renal substance, and containing cheesy matter and pus. The ureter is very liable to become plugged. One or both kidneys may be affected. If the patient lives long enough, mischief of a similar kind almost invariably extends into the lower urinary passages.

The symptoms are few and not characteristic. Pain and tenderness in one or both loins are generally present; but a decided tumour is rare. The character of the urine is broadly the same as in pyelitis, and in fact the diagnosis chiefly turns on the co-existence of pyelitis with evidence of tubercular disease elsewhere. Of course while the ureter is plugged, the urine is quite normal provided the lower urinary passages are unaffected.

The duration varies greatly, but a fatal result is the rule, and generally as the result of phthisis, acute general tuberculosis, or some complication.

RENAL ENTOZOA.

Entozoa are rarely met with in the urinary organs in this country; and *hydatid cysts of the kidney* alone call for some brief comment. The general characters and mode of development of hydatids have already been described as they affect the liver. In the kidney they give rise to a painless, soft, fluctuating tumour in the loin, unaccompanied as a rule with any changes in the urine. Should, however, the cyst burst into the pelvis of the organ, their contents may be discharged with symptoms of "renal colic," or become permanently impacted in the ureter. Suppuration of the kidney may occur from inflammation of the sac. The *treatment* resembles that of hydatids of the liver.

RENAL CALCULUS.

Uric acid and oxalate of lime are not unfrequently deposited from the urine in the secreting tubules of the kidneys, in the form of minute concretions. These may remain lodged in its substance, but more frequently they are washed on into the calices and pelvis of the organ, remaining there and growing by progressive deposit. As to shape, size, and number, they vary greatly. They are apt to become large, and moulded to the cavities in which they lie; occurring singly or in great numbers. Pyelitis, pyo- and hydro-nephrosis are frequent sequelæ of renal calculus, by reason of irritation and obstruction of the urinary passages. When the urine from any cause becomes ammoniacal, a secondary deposit of mixed phosphates is thrown down, and the calculus thus becomes covered with a thick phosphatic incrustation.

Renal calculus may occasion no symptoms of any kind. More often, however, there are darting pains, or aching in one or other lumbar region, radiating to the groin and thigh of the same side; with retraction of the testicle, faintness, nausea or vomiting, irritation of the end of the penis and frequent micturition. These symptoms recur from time to time in a paroxysmal form, and are especially induced by any sudden movement or effort which displaces the calculus. The most severe attacks are met with when the stone enters or passes along the ureter. The sudden onset of intense lumbar radiating pains, vomiting, collapse, or even general convulsions is very characteristic. Usually, after a few hours the attack ceases as suddenly as it began, by the arrival of the stone in the bladder, where it may either remain or be presently discharged by the urethra. Less often the stone becomes impacted in the ureter, and in that case the attack lasts longer, subsides more slowly, and may be followed by pyo- or hydro-nephrosis. The urine in cases of renal calculus is apt from time to time to contain blood, pus, and epithelium from the urinary passages, together with crystalline deposits corresponding to the nature of the concretion.

Treatment.—During the paroxysms of renal colic, warm baths and the free administration of opium are mainly indicated. Belladonna is recommended where opium disagrees.

During the intervals between the paroxysms, the treatment will vary according to circumstances. If there be reason to think that the concretion is composed of uric acid, much benefit may be anticipated from the solvent effects of alkalinised urine. For this purpose citrate or acetate of potash should be given in doses of ℈ii.-ʒi. repeated every three or four hours, and continued for a long period. Oxalate of lime concretions cannot be dissolved in this way; general tonic treatment, a plain bland dietary and avoidance of pastry and sweets are mainly indicated. The removal of renal calculi by surgical interference is an operation which has recently received a good deal of attention, and deserves consideration in all severe cases.

CHYLURIA.

Chyluria, so-called from the chylous or milky condition of the urine, is a rare affection in this country, occurring most frequently in adult females. It is characterised by the discharge of milky urine, which on standing coagulates into a tremulous jelly, an event which sometimes occurs before leaving the bladder. The urine contains a large quantity of molecular fat which is readily soluble in and separable by ether, with fibrin, albumen, and often a little blood. There are no casts. Sometimes the fat is quite absent, while the other conditions persist. The urine is then no longer milky, though it still clots and contains albumen.

The source of this impregnation of the urine with chyle or lymph is not clear, but there appears to be some direct communication in such cases between the lymphatic and urinary systems, which some have ascribed, though on questionable grounds, to the ravages of a small nematoid worm (*Filaria sanguinis hominis*).

No form of treatment is attended with much benefit, but the disorder is not inconsistent with apparently good health, and even a long life.

PAROXYSMAL HÆMATINURIA.

This is an uncommon disease and is almost always met with in connection with exposure to cold. It occurs most frequently in adult males.

It is characterised by the occurrence at irregular intervals of paroxysmal attacks, ushered in abruptly with severe chills. The temperature is *lower* than normal, with a subjective sensation of cold, general malaise, yawning, and nausea. This is shortly followed by the discharge of dark, porter-coloured urine, which deposits an abundant sediment consisting of granular matter (disintegrated blood) with few—if any—blood-cells, oxalate of lime crystals, and granular casts. The symptoms afterwards speedily abate, unaccompanied with sweating, and the urine which is next passed may be normal, or nearly so.

The periodicity of recurrence is regular in some cases, while in others it is very irregular. The length of intermission varies greatly, and relapse is common on the slightest exposure to cold. The pathology is extremely obscure.

As to *treatment*—during the paroxysm the patient should be placed in bed and kept warm, and at all times he should be warmly clad, and carefully avoid any exposure to draughts. The results of medicinal remedies are not very satisfactory, but the administration of quinine, arsenic, or iron has been recommended.

SUPPRESSION OF URINE.

Suppression of urine may depend on a direct suspension of the secretory functions of the kidney *(functional suppression)*; or on the other hand it may be due to some mechanical obstacle to the outflow of urine *(obstructive suppression)*.

Functional suppression of urine is often met with in the algide stage of cholera, in many specific fevers, and in acute Bright's disease. It also occurs sometimes from an over-dose of certain drugs, such as turpentine or corrosive sublimate; or as the result of general shock, especially after injuries of the urethra. The suppression is frequently incomplete; and in this case the scanty urine which is passed, is high-coloured, and contains albumen, blood, and casts. The course is rapid, ending in death or recovery before very long. Death is perhaps more often due to the general condition, of which the suppression is a part, than to the direct effects of arrested secretion.

The best treatment is immediate immersion in a hot bath, with hot gruel enemata, and hot drinks if the patient can swallow and retain them.

Obstructive suppression of urine generally arises from impaction of a stone in one ureter, when the other has previously become blocked. Cancer of the bladder may sometimes block up the lower orifices of both ureters. Strange to say, no symptoms of any importance occur during the first week, but about this time trembling and muscular weakness, contraction of the pupils, slow laboured respiration, dryness of the mouth and complete anorexia supervene. Death usually takes place at the end of two or three days from the first appearance of symptoms. In most cases a little urine is passed from time to time, which is pale, watery, and devoid of albumen and casts. Obstructive suppression, dependent on urethral stricture, is a purely surgical affection.

As to treatment we can do but little. If there be much pain we may employ the treatment already recommended for renal colic. Mechanical means such as shampooing, or kneading the abdomen, have been strongly recommended. With improved methods of operating it may become possible to entertain the idea of surgical interference.

DIABETES INSIPIDUS.

The essential feature of this condition is a substantial and persistent increase in the amount of urine excreted. Recent writers recognise two groups of cases. In the one—termed *Hydruria*—there may be a decrease in the amount of urinary solids, but not necessarily so; while in the other—termed *Polyuria*—there is an increase of the amount of urea, and often also of other urinary solids.

Hydruria.—This affection appears to be confined to the first half of life, and is not uncommon in very young children. It is sometimes hereditary. When acquired, it seems to be related to some kind of nervous disturbance, and may sometimes be traced to cerebral disease or injury, sudden chills, shock, hysterical affections, and the like. The average daily quantity varies from ten to fifteen pints, with a specific gravity of about 1003, of neutral or feebly acid reaction, and in exceptional cases it may contain a little albumen or even sugar. The subjects of hydruria often enjoy fairly good health; but in other cases there are complaints of debility, chilliness, sinking feelings, and irregular action of the bowels. The course is always tedious and permanent cure is rare. Ergot, valerian, strychnia and general tonics have been recommended.

Polyuria.—The etiology of this condition is very obscure. The urinary increase is not so marked as in hydruria, the average daily quantity ranging from five to seven pints. The amount of urea and other urinary solids is considerably increased in typical cases. The specific gravity ranges from 1010 to 1025, the reaction is acid, and there is no albumen or sugar. The usual symptoms are debility, lassitude, erratic pains in the loins and thighs, thirst and constipation. Its course is tedious, and it may prove antecedent to some grave constitutional mischief, and it is also said to occasionally merge into true saccharine diabetes. Opium or codeia appears to be most suitable, associated with or followed up by a course of tonics and cod-liver oil.

CYSTITIS.

Cystitis or inflammation of the bladder may arise from a variety of causes, but the form which occurs in connection with certain diseases of the spinal cord is of most interest to the physician. In some cases the inflammation may be attributed to retention and decomposition of the urine from paralysis of the bladder, but more often it is the result of trophic disturbance directly dependent upon the nervous lesion.

The mucous lining of the organ presents the usual appearances of acute or chronic catarrh, attended with more or less abundant secretion of mucus or muco-pus, and rapid ammoniacal decomposition of the urine.

The main *symptoms* are, pain and tenderness at the hypogastrium and adjacent parts, frequent scalding micturition, and the discharge of urine, which contains more or less mucus or muco-pus. If the urine be ammoniacal, it is apt to become very offensive and of a ropy consistence. Cystitis may be either acute or chronic, and when acute is attended with considerable febrile and constitutional disturbance.

The *treatment* of the acute form consists of hot hip-baths and fomentations, careful regulation of the bowels, a light unstimulating diet, with suppositories of opium and belladonna. Great relief is often obtained by washing out the bladder from time to time with saturated solution of boracic acid, or weak solutions of quinine or hydrarg. perchlor. (1 in 5000). When the urine is retained it must be drawn off. It is obviously of the first importance to discover and remove the cause when possible. Similar principles are applicable to the chronic form.

SECTION VIII.

DISEASES OF THE NERVOUS SYSTEM.

SYMPTOMATOLOGY.

1. GENERAL STRUCTURE AND FUNCTIONS OF THE NERVOUS SYSTEM.

THE fundamental principle underlying the structure of the nervous system consists in the association of nerve-cells or centres with nerve-fibres. The nervous apparatus of man consists of two sets of centres and fibres which are closely related to each other, namely, the *cerebro-spinal* and the *sympathetic* systems. The former includes the brain and spinal cord with the cranial and spinal nerves, while the latter comprises the chain of sympathetic ganglia and the nerves in connection with them.

For the minute structure of the nervous apparatus the student must be referred to anatomical text-books, but some few general considerations are indispensable to the proper study of nervous diseases.

Cerebro-spinal system.—Since nerve-cells alone are capable of exhibiting independent nervous activity, whether in the shape of receiving impressions or of generating nervous force, it follows that true physiological nerve-centres are exclusively confined to the gray matter of the brain and cord. The white fibres on the other hand, wherever they are found, merely serve to conduct impulses to and from the nerve-centres in connection with them.

In the gray matter of the cerebral convolutions we find the centres for volitional movement, conscious sensation, and intellectual activity. The middle convolutions in the neighbourhood of the fissure of Rolando constitute the *motor area.* Irritation of these centres gives rise to definite combined movements; while conversely, destructive lesions are followed by paralysis of corresponding muscular groups. The cerebral convolutions which are *posterior* to the motor area are chiefly concerned with conscious sensation of all kinds, while those which are *anterior* to the motor area are similarly related to volition and ideation.

The white substance underlying the convolutions is made up of nerve-fibres passing in various directions, but for the most part they converge towards the base of the brain. Those from the "motor area" deserve especial attention, as they form the path along which motor impulses travel downwards, from the brain to the muscles. Starting from the convolutions these motor fibres converge to the band of white matter, intervening between the lenticular and caudate nuclei of the corpus striatum (*internal capsule*), forming, however, only the middle third of this band. Thence the fibres pass through the *crusta* of the *crus cerebri*, and on arriving at the medulla mainly cross over to the opposite side with the anterior pyramids, passing down in the antero-lateral columns of the cord to the gray matter of the anterior cornea, and out by the anterior roots of the spinal nerves. The course thus traced constitutes what is called the "motor tract;" and it should be observed —contrary to what was formerly supposed—that it has no direct connection with the corpora striata.

With regard to the "sensory tract" we know much less. Sensory impressions enter by the posterior roots of the spinal nerves, cross over soon to the opposite side, and are conducted upwards to the medulla mainly in the gray matter of that side. Thence sensory fibres pass up to form the posterior third of the internal capsule, and onwards to the sensory cortical centres of the brain. Hence it follows that destruction of one side of the brain causes loss of motion and sensation on the *opposite* side of the body; while destruction of one side of the cord (below the medulla) causes loss of motion on the same side as the lesion, with loss of sensation on the opposite side.

At the base of the brain, we find a series of ganglionic masses of gray matter, some of which are of large size such as the corpora striata, optic thalami, and the corpora quadrigemina; and passing down thence to the termination of the cord below, are several smaller ganglia closely succeeding and connected with each other. Each of these centres is concerned with the reception of different sorts of afferent impressions, or is capable of generating some kind of efferent impulse. The corpora striata have no direct connection either with the motor tract or with the cerebral convolutions, but on the other hand they have an extensive connection with the cerebellum of the opposite side. Provided a lesion of these ganglia does not implicate the internal capsule, there are no symptoms. The optic thalami receive fibres from the cerebellum, the sensory tract, and perhaps from the optic nerves; and from them fibres pass in all directions to the convolutions. Their functions are not known. With regard to the facts which are known relating to other cerebral centres, the reader must consult one of the advanced text-books on nervous diseases.

The *sympathetic system* is so closely related to the cerebro-spinal system both from an anatomical and physiological point of view, that it is difficult to consider it separately. While we have in the sympathetic ganglia, centres which preside over the organic functions of the parts with which they are in connection, it would seem that in the medulla oblongata there is a supreme controlling centre for the whole sympathetic system operating through the communicating branches of the latter with the spinal nerves. The most important function of this system arises from its relation to unstriped muscular tissue, which contracts on stimulation of the sympathetic nerves by which it is supplied, while section of the latter is followed by its relaxation. In this way the length and calibre of the blood-vessels, and therefore the amount of blood supplied to various parts, may be regulated. Apart from the *indirect* influence thus brought to bear upon tissue-nutrition by the sympathetic nervous system, there is reason to think that it may also exert a *direct* influence on the functional activity of the cells in secreting glands and elsewhere.

2. Disturbances of Nutrition.

Though we have yet much to learn with regard to the influence of the nervous system on nutrition, it would appear that tissue-nutrition is largely under the control of certain trophic ganglionic centres, damage to which, or separation from which, is followed by changes in the tissues under their influence. These changes are partly degenerative and partly proliferative in their nature. The following are the most important:—

1. *Peripheral nerves.*—When a nerve is divided, or otherwise badly injured, changes rapidly ensue in both the efferent and afferent fibres on the distal side of the injury. The medullary substance coagulates, and breaks up into drops, which in course of time disappear by absorption. The axis-cylinders are also destroyed, but more slowly. The nuclei of the neurilemma proliferate and become converted into connective tissue (*sclerosis*).

2. *Secondary nervous degenerations.*—The nutrition of the motor tract in the brain and cord depends upon the integrity of its connection with trophic centres in its upper end; while the nutrition of the sensory tract depends on its connection with trophic centres at its lower end. Thus, if the motor tract be interrupted by disease or injury at any part, degeneration of the motor nerve-fibres, together with connective-tissue new-formation (*sclerosis*), extends downwards from the point of interruption (*descending degeneration*). Similarly if the sensory tract be interrupted, sclerosis of the same extends upwards from the point of interruption (*ascending degeneration*).

3. *Muscles.*—In addition to the changes in peripheral nerves above described, rapid atrophy, with loss of electric contractility of voluntary muscles also takes place, when either (*a*) the nerves supplied to them are damaged (*e.g.*, the rapid atrophy of the deltoid muscle after dislocation of the shoulder, attended with stretching, bruising, or laceration of the circumflex nerve); or (*b*) when there is disease of the *anterior* cornua of the cord (*e.g.*, progressive muscular atrophy, and infantile paralysis).

4. *Joints.*—Central nervous lesions similar to those just described may lead to inflammatory changes in the joints—especially in the knees—resembling certain forms of rheumatism, but distinguished from the latter by the paralysis and atrophy of adjoining muscles which usually occur.

5. *Skin.*—Analogous lesions of *sensory* nerves, or of the *posterior* gray cornua of the cord, or sometimes even of the brain itself, may be followed by inflammatory affections of the skin (*e.g.*, herpes zoster in neuralgia, acute sacral bed-sore in acute central myelitis, and acute bed-sore of the buttock in certain cases of cerebral hæmorrhage).

6. *Viscera.*—Certain visceral lesions, such as acute inflammation of the bladder and kidneys, with the discharge of purulent bloody ammoniacal urine, which take place in the course of certain spinal diseases, may probably be explained in a similar way, and quite independently of retention of urine with decomposition.

3. Disturbances of Sensation.

Hyperæsthesia signifies an exalted irritability of the recipient nerve-centres, by reason of which the effects of sense impressions are exaggerated and painful. When the skin is thus affected, the slightest touch causes pain, and similarly the eye may be unable to bear bright light, or the ear loud sounds, without much distress. It is of common occurrence in hysteria, and in certain inflammatory and other affections of the brain and cord. Hyperæsthesia, it will be seen, is quite different from increased delicacy of sensation.

Dysæsthesia or perverted sensibility includes such sensations as tingling, numbness and itching; burning, stabbing, shooting, and other pains; and singing noises in the ears, sparks of light in the eyes, &c.

Anæsthesia denotes an impairment or loss of sensibility which may be either superficial, deep-seated, or involve the whole thickness of a part. Its degree and extent vary greatly. It is often attended with numbness, tingling, or pricking sensations. Sensibility to variations in temperature and muscular sense are not necessarily lost with tactile sensibility. Anæsthesia may arise from organic disease of the brain, cord, or nerves; or from some kinds of functional nervous disease such as hysteria.

General anæsthesia only occurs in connection with general paralysis of the insane.

Hemi-anæsthesia signifies a loss of sensibility over one side of the body. In its complete form (including the loss on one side not only of *common* but also of *special* sensation) it is always of functional origin, and is associated with the hysterical condition. It is further characterised—according to Charcot—by great tenderness over one ovary. As affecting common sensation only, hemi-anæsthesia sometimes occurs in lesions of the brain, affecting the posterior third of the internal capsule, or the parts in direct connection with it. In such cases, it is almost always associated with hemiplegia.

Anæsthesia may also arise from diseased conditions of the pons, medulla, spinal cord, and peripheral nerves. Under these circumstances it is, for obvious reasons, usually associated with motor paralysis.

4. Convulsion—Muscular Spasm.

Under this head are included all abnormal contractions of the muscles, which are either out of proportion to the intensity of an external stimulus, or are caused by some internal pathological irritation. Such contractions are termed *clonic* when they are intermittent, and *tonic* when persistent.

1. *Tremor* is the mildest form of clonic muscular spasm, and consists of a series of slight rhythmical contractions of one or more muscles. Sometimes it is even confined to particular bundles of muscular fibres. Tremor is of two kinds. In the one it persists during repose, and is supposed to be caused by irritation of the nerve-centres concerned. In the other it only occurs on attempting to execute voluntary movements, and is attributed to nervous depression and debility.

Tremor is a prominent symptom of paralysis agitans, disseminated sclerosis, chronic poisoning by lead, alcohol, or mercury, excessive muscular exertion, masturbation, &c.

2. *Choreic movements* resemble simple tremor in ceasing during sleep, and being aggravated by attempts at voluntary movement. At the same time they differ from the irregular movements of true convulsions, in their tendency to affect muscles more or less according to their physiological grouping, and in being moulded, as it were, to definite purposive movements. They vary in degree from simple twitching to violent contortions. In some cases they are limited to a single limb, or to one-half of the body.

3. *Convulsions* consist of irregular energetic clonic muscular contractions, attended with corresponding movements of the affected part. They occur sometimes as the result of the causes acting *directly* on the nervous centres concerned, as in uræmia, epilepsy, and hysteria, or from *reflex* irritation, as in teething, worms, passage of a gall-stone, and the like. Convulsions present every variety of distribution, and sometimes involve the whole body (*general convulsions*). The term *eclampsia* is applied to convulsion which is secondary to some obvious cause, as contrasted with essential idiopathic convulsions, like epilepsy.

4. *Tonic spasm* consists of a persistent contraction involving one or more muscles. The simplest form of tonic spasm is exhibited in "*cramp*." Cramp is a common symptom of certain intestinal disorders, and forms the characteristic feature of tetanus and strychnia poisoning. When a group of muscles is paralysed, their antagonists are apt to become permanently contracted. A third important variety of tonic spasm is met with as a result of abnormal innervation of the affected muscles, as in "spastic paralysis."

5. PARALYSIS.

Paralysis is the opposite of spasm, and denotes an impairment (*paresis*) or loss of motor power. As applied to voluntary muscles it implies an impairment or loss of the power of the will to determine movement. Paralysis may be due to disease of the brain, disease of the spinal cord, or disease of peripheral nerves.

Cerebral paralysis sometimes follows an attack of convulsions, or is caused by injury or disease of convolutions in the motor area. Much more commonly, however, it results from a lesion of the middle third of the internal capsule due to embolism, thrombosis, or rupture of the middle cerebral artery. Paralysis of this kind usually affects the muscles of one side of the body, and is termed *hemiplegia*. In ordinary hemiplegia, the paralysis occurs on the side which is opposite to that on which the lesion exists. The whole of the muscles on that side are not involved. The arm, leg, lower part of the face, and tongue alone are paralysed, whereas the muscles of the abdomen and thorax, certain of the facial muscles (notably the *orbicularis palpebrarum*) the muscles of the eyeball, and those supplied by the eighth nerve of Willis are unaffected. This partial distribution will be understood when we reflect that the muscles which suffer are those which are most under voluntary control, and capable of independent movement; while the muscles that escape are those, which, as a rule, act co-ordinately with the corresponding muscles of the opposite side, and whose combined action the will can only imperfectly restrain. In severe cases, the arm and leg are equally motionless; but it is worth noting that if there is a difference between them, the leg is generally the less affected, and the earlier to recover.

The angle of the mouth is drawn upwards and to the sound side, the cheek hangs loosely, the lower lines of expression are ill-marked, there is difficulty in pursing the lips, and the tongue when protruded diverges to the paralysed side. Owing to the weakness of the tongue and lips the utterance is often defective; but this must not be confounded with the aphasia which so frequently accompanies right hemiplegia. The head and eyes often *deviate* in such cases *to the sound side.* Hemiplegia from lesions of the motor tract *below* the internal capsule is characterised by its frequent association with paralysis of special cranial nerves, the nuclei of which are directly involved. Hence it is that in lesions of the *crus cerebri*, the *pons*, or the *medulla*, cross paralysis is so common—*i.e.*, a paralysed state of *one* side of the body, with that of some cranial nerve on the *opposite side.* Lower down, where the motor tracts of each side approach each other, a lesion may cause paralysis of *both sides* of the body; and from the vital importance of the nerve-centres in this locality, special danger attaches to paralysis of this kind. *Functional hemiplegia* generally occurs in association with hysteria, and is quite independent of any organic lesion. It will be again referred to in the description of hysterical phenomena.

Paralysis due to lesions of the spinal cord almost always affects all those muscles which are supplied by nerves below the upper level of the lesion. Such paralysis is termed *paraplegia.* When the whole thickness of the cord is affected, loss of sensation accompanies the loss of motion. Also, since the centres of micturition and defæcation are seated in the spinal cord, paraplegia is frequently associated with retention or incontinence of urine or fæces. The extent to which the lower limbs are paralysed is often unequal on the two sides; and sometimes only one limb is affected (hemi-paraplegia). The muscles of the trunk, and more rarely those of the upper limbs, may also be affected, according to the level of the spinal lesion.

Paralysis due to injury or disease of peripheral nerves is easily recognised by the fact that the loss of power is limited to the muscles supplied by the nerve in question. It is obvious that such paralysis may present endless variety of distribution. It is further characterised by rapid atrophy and important electrical changes in the affected muscles.

6. CONDITION OF PARALYSED MUSCLES.

Tone.—It is necessary in the first place to distinguish between the results of paralysis which arises from a lesion of the central organs (*central paralysis*) and of that which is due to a lesion of a motor nerve or of the nucleus from which it springs (*peripheral paralysis*). In most cases of central paralysis the muscles preserve their normal tonicity; but in certain cases, especially where the lesion is suddenly produced and extensive, they may at first be limp and flaccid. Sometimes again we meet with muscular rigidity and contraction; but "early rigidity" must be distinguished from that which develops at a later period in the history of the case. "Early rigidity" depends upon irritation at the seat of disease, and is especially common in disease of the membranes of the brain and cord, and in hæmorrhage into the ventricles of the brain. "Late rigidity," on the other hand, comeson in cases of old paralysis, and is due to secondary fibroid degeneration of the motor tract in the spinal cord. In peripheral paralysis, the muscles soon become flaccid and atrophied.

Electrical reaction.—This may be ascertained in two ways:—(1) By *Faradisation*, in which the currents are of momentary duration and of high tension; or (2) by *Galvanism*, in which they are continuous and of comparatively low tension. In the majority of cases of "central paralysis" the electrical contractility is in the first instance quite unaffected; but in some few cases it is exalted, while in others it is impaired. In all such cases the muscles tend very slowly to undergo slight atrophy from disuse, with a loss of electrical contractility *in direct proportion to the amount of atrophy.* In "peripheral paralysis" the muscles quickly atrophy, while their electrical behaviour is peculiar and characteristic. For about the first week the reaction of the affected muscles both to faradism and galvanism becomes diminished—much more rapidly, however, in the case of the former than the latter. From the second to the fourth week all response to faradism is lost, while the galvanic irritability is increased, that is to say, contraction is elicited with fewer cells than before. This state of things is maintained for a few weeks, when at last galvanic irritability gradually passes off, and by that time muscular action is irretrievably lost. There are also certain *qualitative* alterations in the behaviour of the galvanic current. This series of electrical phenomena is known as the "reaction of degeneration."

Electro-sensibility is usually increased or diminished with electro-contractility; but occasionally, as in hysteria, the former may be lost while the latter remains; and more rarely still the reverse is the case.

7. REFLEX PHENOMENA.

Reflex irritability in the majority of cases of "central paralysis" is unimpaired. But in lesions of the spinal cord which cut off all connection between the paralysed part and the brain it is markedly increased. This is also the case in certain irritative lesions of the central nervous system. In "peripheral paralysis," for obvious reasons, reflex irritability is abolished.

Since the spinal cord forms, with the afferent and efferent nerves attached to it, a superimposed series of reflex arcs, it follows that where disease of the nerves can be excluded, observation of the behaviour of such reflexes will yield valuable information as to the condition of the cord segments with which they are in connection. These reflexes may be divided into two groups—*superficial* and *deep*.

The most important *superficial* reflexes are (1) the *plantar reflex*—movements of the leg obtained by tickling the sole of the foot; (2) *cremasteric reflex*—withdrawal of the testicle on stimulation of the inner side of the thigh; (3) *abdominal reflex*—by irritation of the abdomen at outer edge of the rectus muscle; and (4) *epigastric reflex*—by scratching the skin over the lower rib cartilages. These reflexes are diminished or abolished by disease of the segment of the cord with which the nerves concerned are connected, and are exaggerated by any lesion which removes such segments from cerebral control, as by a transverse lesion of the cord above the segment concerned.

The most important *deep reflexes* are the *knee-jerk*, and *ankle-clonus*. The *knee-jerk* is elicited in health by sharply striking the patellar tendon with some blunt object, while the leg hangs loosely from the knee. The afferent and efferent fibres concerned in it join the cord with the second and third lumbar nerves. If this reflex arc be interrupted at any point, by disease of the cord or otherwise, the phenomenon is absent, as in locomotor ataxy. On the other hand, when the arc is cut off from the inhibitory influence of the brain by a lesion higher up in the cord, as in transverse myelitis, it is exaggerated.

Ankle-clonus is characterised by a persistent series of rapid rhythmical movements of the foot at the ankle-joint, on sudden and forcible passive flexion of the latter on the leg. It cannot be obtained in health, but under similar conditions to those in which the knee-jerk is exaggerated, it may be readily elicited. The primary source of this reflex is a stimulus afforded by sudden tension of the muscles of the calf. The reflex centre is situated in the cord at the level of the first and second sacral nerves.

8. APHASIA.

Aphasia denotes an inability to express thoughts by means of written or spoken language. On the one hand, it must be distinguished from mental incapacity or absence of ideas; and, on the other hand, from defects in the apparatus of articulation.

The most common and typical form of aphasia arises from inability to co-ordinate the motor mechanisms concerned in spoken or written language (*efferent* or *motor aphasia*). This faculty is situated in the hinder ends of the second and third left frontal convolutions of the brain, which, together with the closely adjoining internal capsule is supplied by the middle cerebral artery. These facts explain at a glance the relationship which so often occurs between aphasia and right hemiplegia, caused by extensive extravasation around the left internal capsule, or by embolism or thrombosis of the left middle cerebral artery. But while the association of aphasia and right hemiplegia forms the rule, aphasia may occur independently of hemiplegia, either from a lesion limited to the convolutions mentioned, or from a temporary suspension of their function in common with those of other parts of the brain. In left-handed people aphasia is associated with left hemiplegia.

In a typical case of motor aphasia the patient's mind is intact, he understands what is said to him, and apparently what he reads, but he is either altogether unable to express his ideas by speech or writing, or does so incorrectly and with difficulty. Automatic words and phrases which involve no intellectual process may be expressed easily, and often replace the word which is really wanted. Thus a reply to any question may be limited to the words "yes" or "no." As a rule the patient is fully aware of the mistakes he makes, though he cannot avoid them.

There is another form of aphasia (*sensory aphasia*), where the patient fails to understand spoken words though he can hear them perfectly well (*word-deafness*); or fails to understand written or printed words, though he can see them perfectly well (*word-blindness*). The former is due to a lesion in the upper end of the first temporo-sphenoidal convolution (left), while the latter is due to a lesion of the parieto-occipital convolution (left). Associated with sensory aphasia, there is always more or less loss of memory of words (*amnesia*), and the intelligence is generally impaired.

9. HEADACHE.

Headache is not only one of the commonest, but often one of the most distressing symptoms of cerebral disorder. The pain is variously described as dull, heavy, shooting, throbbing, constricting, &c. It may be either more or less widely diffused, or localised to a particular spot, or confined to the course of some sensory nerve. As to its seat it may be general, frontal, vertical, occipital, or one-sided.

Cases of headache may be arranged in two groups: (1) *cranial headache*—due to some disorder of the cranial structures, *e.g.*, neuralgia of the scalp, osteitis, or periostitis; and (2) *cerebral headache*—arising either from inflammation or organic disease of the brain, or of the membranes which cover it; or from some alteration in the quality or quantity of the blood supplied to the brain. In the latter case it often occurs in connection with affections of remote parts.

In practice, it is frequently a matter of great difficulty to relegate a headache to its precise cause. When the pain is severe, constant, and localised, disease of the bone or subjacent membranes may be indicated. In neuralgia of the scalp the pain is superficial, paroxysmal, associated with tender points, and almost always one-sided, since its distribution corresponds with that of a sensory nerve. Headache from structural diseases of the brain is usually severe, persistent (though sometimes paroxysmal) aggravated by the recumbent posture, and often associated with convulsive or paralytic symptoms. In doubtful cases, valuable information may be gained by the use of the ophthalmoscope, showing the presence or absence of inflammatory changes in the optic disc or retina. Headache from gastro-hepatic disorder is generally frontal, while that of anæmia and exhaustion is vertical and often associated with a feeling of burning and pressure at the same spot. Congestive headaches are aggravated on lying down or stooping, while the reverse obtains in those dependent upon anæmia. It is also worth bearing in mind that headache is very frequently set up by carious teeth and refractive defects of vision, attention to which will often lead to the cure of cases which have resisted all general treatment.

10. Delirium.

Delirium is an acute disorder of the mind, which varies from mere incoherence of ideas to the most complete mental derangement. It may be mild and quiet, busy and garrulous, low and muttering, or wildly maniacal. It depends proximately upon irritation of the psychical centres of the brain. The circumstances under which it occurs deserve attentive consideration, on account of the indications thus afforded for appropriate treatment. It usually arises from one of the following causes:—(*a*) inflammation or organic disease of the brain; (*b*) retention in the blood of histolytic products from interference with elimination; (*c*) high temperature; (*d*) alcoholism; or (*e*) violent mental shock. It must be borne in mind that liability to delirium, its character, and intensity are largely dependent on the general nervous constitution of the patient.

11. Vertigo.

Vertigo or giddiness implies a feeling of instability associated with involuntary movement of the body, or a sensation of movement either of the body or of surrounding objects. It implies a disorderly action of the nervous centres concerned in maintaining equilibrium, of which probably the cerebellum is the most important.

Vertigo may be produced by direct disturbance of the equilibrating centres as the result of organic disease, sudden variations in the blood-supply, slowness on the part of the vascular walls to adapt themselves to sudden alterations in posture, or of toxic causes (*e g.*, tobacco). On the other hand it may be due to some interference with the centripetal impressions from the eye, ear, muscles, or skin, which regulate the action of the co-ordinating centres. Lastly, we meet with vertigo as an essential feature of mild epilepsy, or forming the "aura" of severe epileptic attacks.

From most of these causes, vertigo does not amount to more than a vague feeling of unsteadiness. Sometimes, however, it is distinctly paroxysmal and definite (the movement or sense of movement being constantly in one definite direction), and in such cases is almost always due to aural disturbance (labyrinthine vertigo) or epilepsy.

Aural or labyrinthine vertigo (Menière's disease) may result from any aural disorder which directly or indirectly affects the labyrinth of the internal ear. It is characterised by tinnitus, deafness, vertigo, and is frequently associated with nausea or vomiting. The attacks though often severe are not dangerous. The paroxysm gradually passes off, but some tinnitus, giddiness and disturbance of the stomach generally persist for a few days. Recurrence is frequent; but, after a while, the symptoms usually disappear, with the exception of deafness, which is commonly permanent.

Epileptic may be distinguished from aural vertigo by loss of consciousness, and quick complete recovery. There is no giddiness during the intervals, and there is no persistent tinnitus. If accompanied with convulsive movements, of course the diagnosis is easy.

12. INSENSIBILITY—STUPOR—COMA.

These terms are used to express different degrees of functional incapacity, beginning at the cerebrum, and characterised at the outset by loss of consciousness, perception, and voluntary motion. In this stage, automatic and reflex movements continue. Afterwards the large ganglionic masses at the base of the brain become involved, and the capacity for automatic movements becomes more and more limited. In still more serious cases the loss of function extends to the medulla (which is the seat of the centres presiding over the important functions of circulation and respiration amongst others), and death ensues.

Pathologically, stupor and coma may be induced by disturbance of the cerebral circulation (*e.g.*, extreme congestion or anæmia); deficient oxygenation of the blood (*e.g.*, asphyxia); toxic conditions (*e.g.*, uræmia); or by inflammation, structural disease, or injuries of the brain.

SUB-SECTION I.

DISEASES OF THE BRAIN.

CEREBRAL MENINGITIS.

MENINGITIS broadly denotes an inflammation of the meninges; but it rarely happens that all these membranes are simultaneously involved, and therefore special terms are necessary to distinguish inflammation of one membrane from that of another. Thus inflammation of the dura mater is called *pachymeningitis;* of the surface of the arachnoid—*arachnitis;* of the pia-mater and sub-arachnoid tissue—*leptomeningitis.* Now the first two are probably *always* secondary to surgical injury or disease of the skull, and therefore need not occupy consideration here. The simple and tubercular meningitis about to be described are forms of leptomeningitis. Acute simple meningitis, although often secondary to disease or injury of the bones of the skull, also occurs in connection with certain acute specific fevers, erysipelas, conditions of profound debility, nervous shock, or syphilis. In a chronic form it may also be due to syphilis. But of all forms of meningitis, that set up by tuberculosis is the most common, especially during childhood.

ANATOMICAL CHARACTERS.—*Acute simple meningitis* generally affects the *convexity* of the brain. It is characterised at first by acute congestion of the pia-mater, which is soon followed by the formation in the sub-arachnoid space, and in the intervals between the convolutions, of a firm yellowish-green exudation of lymph and inflammatory cells. In this form, the arachnoid cavity and ventricles are generally empty; but when the base of the brain is involved, they are apt to contain an excess of fluid. When occurring as the result of injury, *all* the membranes are involved.

In *chronic meningitis* the membranes are thickened, opaque, and more or less adherent to each other and to the brain substance beneath.

Tubercular meningitis is usually limited to the *base* of the brain, and is characterised by the presence of gray miliary tubercles in the pia-mater, in association with ordinary inflammatory changes consisting of an opaque yellowish exudation in the sub-arachnoid space. These changes are especially abundant around the optic commissure, extending thence into the adjoining fissures of Sylvius, and backwards towards the medulla. As in simple meningitis the arachnoid cavity is empty, but the ventricles are more or less distended with clear or flocculent serum, which leads to flattening of the convolutions by outward pressure against the skull.

The *symptoms* of *acute meningitis* (both simple and tubercular) may be conveniently considered together. *Premonitory symptoms* lasting a very variable time are observed in tubercular meningitis, but are usually absent in the simple form. The patient is fretful, restless, loses his appetite, suffers from a furred tongue, constipation, headache, and feverishness, with a quick and irregular pulse.

The established disease may be divided into three stages.

1. *Stage of irritation.*—The invasion or beginning of this stage is sometimes characterised by rigors or chilliness followed by fever, and intense headache; in other cases by obstinate vomiting, by a strange wild manner, or by an epileptic seizure. Some of the following symptoms now make their appearance.

General.—There is marked fever, often of a very irregular type; a frequent pulse, subject to *sudden variations independently of the fever;* irregular and moaning breathing, nausea and obstinate vomiting, with thirst, loss of appetite, and constipation. *Local.*—Headache is constant, and often so severe that the patient shrieks with the pain, and supports his head with his hands. This is often associated with tenderness of the scalp and radiating pains in the limbs. *Mental.*—The patient is fretful, restless, with a tendency to active delirium. *Motor.*—There may be tremor or twitchings of the face and limbs; strabismus with contracted pupils; general convulsions, or tonic muscular contractions. *Sensory.*—Not infrequently there is also some general hyperæsthesia, with intolerance of light and sound.

2. *Stage of transition.*—The headache and other marked symptoms of the first stage now gradually subside, and the patient becomes for the most part quiet and drowsy. The temperature generally falls, sometimes to normal, or even below it. The *pulse is slow* and irregular; the

respiration is also irregular and sighing. The patient is dull and apathetic; food is only taken when offered, and then often with difficulty. He rolls his head uneasily from side to side, and picks at the bed-clothes. The pupils are dilated; paralysis may supervene, especially ptosis, and paralytic strabismus; and he usually suffers from quiet delirium. At this time some rather characteristic phenomena may be observed; (*a*) a short, sharp, shrill scream occurring at intervals and known as the "*hydrocephalic cry*"; (*b*) *retraction of the abdomen;* (*c*) a red blush which persists after irritation of the skin, known as the *tâche cérébrale;* and (*d*) *optic neuritis.*

3. *Stage of coma.*—Many of the preceding symptoms continue, but this stage is mainly characterised by coma and convulsions. Absolute insensibility prevails, the patient can no longer be roused, and common and special sensation are lost. The pulse once more becomes rapid, but is now very feeble. The breathing is also frequent and irregular. The temperature varies greatly—in some cases it rises rapidly as death approaches, in others it falls below normal. The tongue is brown and coated, and the lips and teeth are covered with sordes. Convulsions recur more and more frequently, and often constitute the closing scene of a disease which is almost always fatal.

It should be borne in mind that numerous departures from the above general description are met with in practice; that the division into stages is by no means always well-marked; that the passage from one stage to another is, as a rule, gradual; and that their absolute and relative duration vary much, the total duration being from one to three weeks, or even longer.

Such differences as may exist between simple and tubercular meningitis are largely due to the fact that the former is generally limited to the *convexity*, and the latter to the *base* of the brain. In the *simple* form the onset is usually sudden; delirium comes on early and is of an active kind; well-marked remissions in the fever are wanting; and the course is rapid. In the *tubercular* form on the other hand, the onset is insidious, irritation followed by paralysis of cranial nerves is a prominent and early feature; delirium does not come on till late, and is then comparatively quiet; there are well-marked remissions in the fever and other symptoms; the temperature in the last stage is low; and the general development of the symptoms is slow.

From *typhoid fever*, to which acute meningitis has some resemblance at the onset, the distinction rests upon the nervous features of the premonitory symptoms, the irregular course of the temperature, the retracted abdomen, and the absence of diarrhœa splenic enlargement and rash in the latter affection.

It is also perhaps necessary to call attention to the similarity of the symptoms sometimes caused by worms or severe diarrhœa in young children, and those of meningitis.

The *symptoms of chronic meningitis* are obscure, and in some respects resemble those of cerebral softening. At the outset they consist of constant headache with indications of cerebral irritation. At a more advanced period the memory is obviously impaired, the mind is feeble, and the gait is tottering. Incomplete paralysis of the limbs, of variable distribution, may supervene.

CEREBRAL CONGESTION AND ANÆMIA.

THE proper functions of the brain (as of all other organs), are closely related to its blood-supply. The symptoms of active cerebral congestion are mainly characterised by *irritation* and *excitement*, and those of mechanical congestion and anæmia by *depression*. At the same time it is not easy—nor, in fact, is it possible—always to descriminate precisely between the symptoms which are due to the circulatory trouble alone, and those which are due to alterations in the quality of the blood and to other conditions with which congestion and anæmia are associated.

Congestion.—Active cerebral congestion may arise from irritation of the brain from any cause, chilling of the surface of the body, exposure to great heat, high fever, and certain toxic agents such as alcohol, nitrite of amyl, &c. It is also a common accompaniment of some functional nervous diseases (*e.g.*, Graves' disease) and the climacteric state in women. Mechanical congestion may be caused by obstruction, diseases of the lungs or heart, or by any local affection which interferes with the return of blood from the brain.

The general *symptoms* of *active* cerebral congestion are —extreme wakefulness, incapacity and disinclination for mental work, confusion of ideas, defective memory, emotional excitability with occasional temporary illusions or hallucinations. Headache and dizziness are prominent symptoms, with a feeling of heat and fulness about the head, flushing of the face and throbbing of the carotids, which is exaggerated on stooping or lying down. Strong light and noise cause annoyance, and sparks before the eyes, singing noises in the ears, with tingling sensations in the limbs may be complained of. There is usually great restlessness with occasional startings and twitchings of the limbs. The pupils are contracted.

Special symptoms are also apt to occur in the course of active cerebral congestion. (1) An *apoplectic attack* is the most frequent and important. The patient becomes giddy, and falls in a condition of partial insensibility, which is followed by paralysis of one or more limbs, or of one side of the body. The paralysis is never complete, rarely involves the facial muscles, and passes off comparatively soon. (2) *Convulsions* may occur, and are the most frequent result of active congestion of the brain in infants, though of course rare in comparison with other causes of infantile convulsions. (3) Acute maniacal attacks are also sometimes met with.

The symptoms of mechanical cerebral congestion are, on the whole, analogous to the above; but dull headache, drowsiness, and tendency to stupor are the most prominent features. Intermittent congestion—such, for instance, as may arise from violent paroxysms of coughing—is attended with more striking symptoms than when the congestion is steadily persistent. Under these circumstances convulsions sometimes take place; but apoplectic and maniacal symptoms are less frequent than in active congestion.

ANÆMIA.—Cerebral anæmia may be due to general anæmia, or again it may be traceable to organic disease (especially aortic regurgitation) or weakness of the heart. The phenomena of syncope depend upon cerebral anæmia. The partial anæmia caused by embolism, thrombosis, &c., is not at present under consideration.

In sudden anæmia of the brain, the main symptoms are drowsiness, giddiness, failure of special senses, singing noises in the ears, dilated pupils, numbness, mental confusion, and nausea. Headache is not common. If more severe, there may be complete insensibility, convulsions, and general collapse, passing into fatal coma. When the anæmia is chronic and persistent, we meet with general mental irritability, restlessness, troublesome insomnia, inaptitude for mental work, loss of memory, and severe general or vertical headache relieved by the recumbent posture.

A special form of cerebral anæmia, occurs in infants, commonly as a sequel to exhausting diarrhœa. It is characterised by drowsiness and general collapse gradually merging into coma, pallor, cold extremities, *depressed* fontanelles, retracted head, and often slight squint. From some resemblance to the symptoms of hydrocephalus it was termed "hydrocephaloid" by Marshall Hall.

ACUTE ENCEPHALITIS.

Encephalitis or inflammation of the brain may be either general or local, and is always of secondary origin. General encephalitis may be caused for instance by general meningitis, or by thickly scattered embolic abscesses, as in pyæmia. Local inflammation is usually secondary to injury or disease of the skull (*especially disease of the internal ear*), or to irritation set up by new growths or extravasated blood, or to pyæmic abscesses.

Anatomical Characters.—The inflamed brain-substance is at first red and congested, afterwards becoming swollen, pulpy, and more or less disorganised (*acute softening or ramollissement*). The surrounding tissue is congested and œdematous.

In course of time the inflammation subsides, or it terminates in suppuration. When occurring as the result of cranial injury or disease, a cerebral abscess is usually single, and occurs in the white substance of the cerebrum. It may be quite small, or it may occupy the whole of one cerebral hemisphere, compressing and flattening the convolutions against the skull. In recent cases, the pus is fairly healthy, and the abscess is only surrounded by softened brain substance; but in more chronic cases it is often enclosed in a firm fibrous capsule containing unhealthy and fœtid pus. Such abscesses usually increase in size till they burst either on the surface of the brain, or into a ventricle, or into the tympanum, or in rare cases externally. The multiple abscesses of pyæmia are usually small, and scattered promiscuously throughout the substance of the brain.

Symptoms.—A systematic description of the symptoms of encephalitis is almost impossible, not only on account of the wide variations according to the seat and extent of the mischief, but on account of the difficulty of isolating the symptoms proper to encephalitis from those of the affection to which it secondary.

In the milder forms of inflammation they are exceedingly vague, and amount to little beyond slight febrile disturbance, headache, giddiness, some exaggeration of any motor or sensory disturbance due to the original lesion, and mental incapacity; and perhaps some delirium, or an intercurrent attack of convulsions or of coma.

Cerebral abscess often runs an entirely latent course, but it may be assumed to have occurred when, after some disease or injury to the head, or in the course of a chronic otorrhœa, inflammatory and febrile symptoms supervene, accompanied with localised headache, vomiting, convulsions or coma, or other cerebral symptoms. Its progress sometimes admits of a division into a stage of irritation, and one of depression such as described under the head of meningitis. Paralysis occurs in about a half of all cases, and usually takes the form of hemiplegia. Cerebral abscess usually proves fatal in from one to four weeks, and death is commonly preceded by coma. In pyæmic abscess death is due to the primary disease, rather than to the cerebral complication.

CEREBRAL HÆMORRHAGE.

Cerebral hæmorrhage (or extravasal apoplexy as it was formerly called) exclusively arises from rupture of a vessel in the brain, and is brought about by one or more of the following conditions:—(1) *Diseases of the vascular walls* such as fatty degeneration, chronic endarteritis, or small aneurismal dilatations, by which their fragility is increased; (2) *increased pressure of the blood* within the vessels, such as occurs in chronic Bright's disease, or as a result of overstrain or prolonged effort; and (3) *any change in the surrounding parts by which the vessels are deprived of their natural support*, such as may be assumed to arise from cerebral softening. Cerebral hæmorrhage is essentially a disease of advanced life, and is more common in men than in women.

Anatomical Characters.—Capillary hæmorrhages consist of a cluster of punctate extravasations over an area of variable extent. When due to the rupture of larger vessels, the hæmorrhage varies in size according to the calibre of the vessel concerned. When of small size the nerve-fibres are merely pushed asunder; but when large, the brain substance is ploughed up, and a potential cavity is formed, of roundish shape, with ragged walls, and filled with blood which soon clots. The hæmorrhage may extend into the ventricles, or, more rarely, through the cortex of the brain into the arachnoid cavity. Usually there is only a single hæmorrhage, which is most frequently seated in or near the lenticular nucleus of the corpus striatum; less frequently in the optic thalamus, or in the mass of a cerebral hemisphere; sometimes in the crus cerebri, pons Variolii, or cerebellum; most rarely in the corpora quadrigemina and medulla.

Should the patient survive, the *clot* undergoes certain changes. It first shrinks, and the serum separates out. It then undergoes disintegration, together with the broken nerve-tissue, and forms a dark-red liquid which first becomes brown and afterwards yellow. Meanwhile, the cavity becomes rounded, and its boundary more defined. As a result of reactive inflammation a fibroid cyst is ultimately formed, the cavity of which is traversed by delicate threads of connective tissue, and contains clear serum. This condition may persist; or the fluid may be absorbed, followed by approximation of the cyst-wall, until a pigmented cicatrix alone remains. Crystals of hæmatoidin are often met with. The secondary effects on the *surrounding tissue*, are:—(1) flattening of the convolutions and cerebral anæmia which at once results from the outward pressure of the clot; (2) local encephalitis due to irritation set up by the clot, which sets in after four or eight days; and (3) at a much later period, fibroid degeneration of the motor tract on the damaged side from above downward (*secondary degeneration*).

Symptoms.—*Premonitory* symptoms are not common, but when present, consist of headache, giddiness, slight hesitation of speech, forgetfulness of words, mental dulness, sensory disturbances (muscæ volitantes, tinnitus aurium, numbness or tingling in the limbs, &c.), weakness of the limbs on one side of the body, or nose-bleeding.

The symptoms of cerebral hæmorrhage may for the most part be included in two groups; (1) *paralysis* which directly depends upon the local injury; and (2) an "*apoplectic stroke*," which is probably due to the sudden functional disturbance of the brain as a whole.

The nature of the paralysis varies according to the extent and the seat of the hæmorrhage. It is obvious that the smaller the extravasation, the less prominent are the symptoms to which it gives rise. Hæmorrhage most frequently occurs from rupture of the lenticulo-striate branch of the middle cerebral artery which lies between the external capsule and the lenticular nucleus of the corpus striatum. From this point the hæmorrhage usually extends inwards and upwards, and involves the motor tract in the anterior two-thirds of the internal capsule. It is therefore attended with paralysis *on the opposite side of the body*, of the arm and leg, of the lower facial muscles, and often of the muscles which protrude the tongue (*hemiplegia*). It is important to note that the eyes can be closed together, and that the rhythmical movements of the thorax and abdomen continue.

So long as the motor tract is not structurally damaged, the paralysis is only temporary; but if otherwise, it is permanent. Hæmorrhage into the left half of the brain, is as a rule, accompanied with *aphasia* (see p. 353). Sometimes *hemi-anæsthesia* is associated with hemiplegia, and this indicates the implication of the hinder part of the internal capsule. More or less rigidity of the paralysed limbs is also frequently observed, especially after extensive hæmorrhage into the corona radiata, or into the ventricular cavity, and these cases are usually characterised by very profound coma.

Hæmorrhage into the *crus cerebri* is characterised by paralysis of the third or fourth cranial nerves on the *same* side, together with hemiplegia—and sometimes hemi-anæsthesia too—on the *opposite* side of the body. Cross paralysis of a similar kind may follow certain cases of hæmorrhage into the *pons*, and the cranial nerves concerned in deglutition and articulation are in this case especially apt to be involved. More often there is general paralysis. Lesions of this part are usually quickly fatal. In hæmorrhage into the *cerebellum*, paralysis is often absent, but on attempting to walk the patient is apt to stagger; and severe pain at the back of the head, vomiting together with vertigo, are also of frequent occurrence.

The "apoplectic stroke" is essentially characterised by coma. The patient more or less suddenly and completely loses consciousness, sensation, and motion. The pulse is slow but full; the breathing is slow, laboured, and stertorous; the cheeks are puffed out at each expiration; the face is usually flushed, and the carotids may throb violently; the eyelids are closed; the eyes—and sometimes the head—are turned to the side opposite to the paralysis, and the pupils are unequal or contracted. Reflex irritability may be abolished at first, but returns in a short time. The sphincters are often temporarily paralysed, leading to involuntary discharge of urine and fæces.

The *clinical history* of cerebral hæmorrhage varies greatly according to its seat, extent, and rapidity of occurrence.

1. Occasionally profound coma supervenes, with or without premonitory symptoms, in which the patient dies in a few minutes or after some hours—*never instantaneously*. This variety is often ushered in with convulsions.

2. More often there is first an apoplectic fit, but after some hours consciousness gradually returns; but hemiplegia with or without aphasia remains, and persists for a long time or for the remainder of life.

3. Partial recovery may occur as before, but owing to fresh hæmorrhage the coma returns and proves fatal.

4. In cases where the hæmorrhage occurs slowly in the first instance, symptoms of paralysis may precede the apoplectic attack, if the latter takes place at all.

5. There may be no apoplectic fit at all, temporary hemiplegia affording the only sign of hæmorrhage having taken place.

There are, in conclusion, certain secondary phenomena which deserve mention. 1. As a result of *local encephalitis* set up by the clot towards the end of the first week, febrile symptoms attended with headache, mental confusion, or even delirium, a tendency to coma, and sometimes convulsive movements may be observed. These symptoms usually continue for four or five days and then pass off. 2. *A peculiar permanent change of manner* with loss of memory and emotional perversion is apt to occur after cerebral hæmorrhage. 3. *Bed-sores* sometimes appear on the buttock of the paralysed side, from the second to the fourth day, and are of very unfavourable augury. 4. *Inflammation of internal organs—e.g.*, the lungs—sometimes supervenes. 5. After the second or third week, or later, rigidity with flexion of the paralysed limbs may occur, accompanied with atrophy of the muscles (*late rigidity*). This is due to secondary descending degeneration in the motor tract of the cord. 6. *Other nervous symptoms* more rarely occur, such as choreiform movements, delirium, mania, or dementia, which are due to secondary changes in the nervous centres.

The prognosis of cerebral hæmorrhage depends upon the seat of the mischief, and the severity of the symptoms. For instance, hæmorrhage into the ventricles, or medulla is always fatal. Speaking generally, cases in which insensibility persists for over twelve hours are always fatal. Cheyne-Stokes' respiration is a very unfavourable sign; and for obvious reasons the co-existence of organic disease in other parts renders recovery less probable.

MENINGEAL HÆMORRHAGE.

Under this head are included hæmorrhage into the subarachnoid space, and into the arachnoid cavity. Traumatic injury is its most common cause, but sometimes it is due to arterial degeneration and rupture, or to the bursting of an aneurism at the base of the brain; and more rarely it is met with in connection with paralysis of the insane.

Symptoms.—Slight hæmorrhage may give rise to no recognisable symptoms whatever. On the other hand if the hæmorrhage be abundant and take place suddenly, the patient is plunged into a state of fatal coma, which cannot easily be distinguished from that due to intracerebral hæmorrhage. Such attacks, however, are very frequently associated with general convulsions. When the onset of hæmorrhage is more gradual, the coma may be preceded by severe headache, giddiness, vomiting, and general or local convulsions. Apart from the general muscular relaxation in severe cases, paralysis is rare. Death may follow in a few hours, but often partial recovery takes place, until a renewal of the bleeding once more plunges the patient into a state of coma. In exceptional cases the onset of coma may be deferred for a considerable period after the original injury. Complete recovery is rare.

Injury in the course of birth sometimes causes meningeal hæmorrhage and accounts for certain cases of so-called "congenital" nervous defects of various kinds.

CEREBRAL EMBOLISM AND THROMBOSIS—ACUTE SOFTENING.

Cerebral embolism is caused by plugging of one of the cerebral arteries—almost always the *left middle cerebral* artery. The embolus usually takes the form of a small vegetation or particle of fibrin which becomes detached from the heart or aorta, and is swept into the current of the circulation. Accordingly it is found most frequently in connection with *valvular disease of the left heart*, and (less often) with degeneration or aneurism of the aorta. Pyæmic abscesses are, strictly speaking, the result of cerebral embolism, but they are not included in the present description.

Cerebral thrombosis is almost always associated with chronic inflammatory, syphilitic, or other disease of the cerebral vessels, which leads to a local coagulation of blood within them. Its frequency increases with age.

Both embolism and thrombosis have this feature in common—that *the supply of blood is cut off* from that part of the brain to which the obstructed vessel is distributed; and that consequently if collateral circulation is not quickly established, and if death does not intervene, *softening* and *necrosis* of the same area necessarily follow. The affected part becomes soft, pulpy, or even diffluent, and is of a yellowish colour, due to altered blood-pigment *(yellow softening)*.* Microscopic examination reveals granulation-corpuscles and nerve-fibres in various stages of fatty degeneration. In softened patches of small size *absorption* may occur, and only a small scar may be left behind; in other cases the absorption is not complete, and a cyst is formed, traversed by loose cellular tissue, and containing a turbid fluid.

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* An undue importance has, in my opinion, been attached to the epithets *red*, *yellow*, and *white*, as applied to varieties of cerebral softening, and it would be well if they were discarded. Those who may still desire to attach some definite meaning to them may regard (1) *red softening* as exclusively arising from *acute inflammation;* (2) *yellow softening* as indicating a *necrotic process* such as that due to *embolism* or *thrombosis;* and (3) *white softening* as that met with in hydrocephalus, which is partly the result of maceration and partly of slow inflammatory change.

Symptoms.—In embolism *premonitory symptoms* are wanting; but in thrombosis, owing to the general arterial disease with which it is associated, premonitory symptoms may be observed very similar to those which sometimes precede cerebral hæmorrhage.

In *embolism* the onset is more or less sudden, and is marked by loss of consciousness which varies greatly in degree, but is rarely altogether wanting. This condition may pass on into that of death, but more commonly consciousness returns, and the patient is then found to be paralysed. Of course the paralysis will vary according to the seat and extent of the obstruction; but as already pointed out, by far the most frequent seat of embolism is the left middle cerebral artery, which supplies the greater part of the corpus striatum, the internal capsule, the anterior half of the optic thalamus, and nearly the whole of the antero-lateral region of the brain. Hence there is usually right hemiplegia with aphasia.

In *thrombosis* a sudden onset is exceptional, and complete coma is rarely observed. Headache, vertigo, mental confusion, and faintness, are common initial symptoms, and *develop gradually*. The paralysis which follows is also characterised by slow and gradual development, while its progress is marked by successive remissions and exacerbations. Convulsive movements sometimes occur in the early stages of thrombosis.

Though the primary attack, whether of embolism or thrombosis, may be survived, the paralysis often persists, accompanied with other indications of cerebral softening such as more or less persistent frontal headache, loss of memory, and general mental enfeeblement passing on to complete dementia. Bed-sores on the buttock are also apt to appear, as after cerebral hæmorrhage. Similarly, "late rigidity" with contractions of the paralysed limbs may occur from secondary lesions of the motor tract in the spinal cord, which may be followed by nutritive lesions in the joints and muscles. The prognosis is therefore very unsatisfactory, death sometimes occurring early from coma, or after a variable interval—from asthenia or some complication; while a few cases completely recover.

TUMOURS OF THE BRAIN AND MEMBRANES.

THE more important of the new formations to which the brain and membranes are liable are as follows:—

1. *Tubercle.*—This constitutes the commonest form of cerebral tumour. They are single or multiple, and consist of rounded cheesy masses, resulting from the degeneration of aggregated groups of tubercle. They vary from the size of a mustard-seed to that of a hen's egg, and sometimes are even still larger. Their usual seat is in the cortical gray matter of the cerebrum or cerebellum.

2. *Glioma.*—This is also a frequent form of cerebral tumour, and arises in connection with the neuroglia, or supporting connective tissue of the brain substance. It consists of fibres and cells, and it is usual to distinguish a hard and soft variety, according to the predominance of fibres and cells respectively. Glioma is closely related to sarcoma, and is similarly liable to undergo mucoid degeneration. It may occur in any part of the brain.

3. *Syphilis.*—Syphilitic gummata generally occupy a superficial position in the cerebral cortex or pons, and as a rule are connected with or adherent to the meninges.

They are almost always multiple, vary much in size, and are accompanied with a good deal of inflammatory change in the parts around, thus leading to caries, meningitis, and encephalitis. When situated at the base of the brain, the cranial nerves are also particularly liable to become involved. In syphilis, the *small arteries* are also subject to a fibro-nuclear infiltration of their inner coat, which seriously narrows their calibre and in this way becomes a cause of thrombosis.

Other kinds of cerebral tumour are sometimes met with, *e g.*, cancer, myxoma, lipoma, hydatids, and aneurism; but on account of their rarity, no special description of them is necessary here.

The frequency of cerebral tumours is twice as great in males as in females, and much greater in childhood and up to forty years of age, than in later years. Tubercular tumours are most common in childhood, and gliomata most common in adult life. With a history of syphilis, syphilitic tumours are perhaps the most frequent.

SYMPTOMS.—First it may happen that even a large tumour may exist without giving rise to any symptoms whatever during life, especially when its growth has been very slow. In other cases a tumour may exist and cause no symptoms until a few days before death, when convulsions occur followed by fatal coma. Lastly, it is obvious that in all cases the symptoms will vary much according to the nature, size, seat and rapidity of growth of the tumour.

1. *Headache* is usually the earliest and most prominent symptom. It is *intense*, and generally *persistent*, though often exhibiting paroxysmal exacerbations. Its character is very variable, and its seat by no means always corresponds with that of the disease. 2. *Vertigo* is rarely absent, and frequently constitutes an early symptom.

3. *Vomiting* is also of common occurrence. It is not preceded by nausea as a rule, is unattended by the signs of gastric derangement, and is generally associated with constipation. 4. *Sensory disturbances.* The *eye* is often the first to suffer. There may be dimness of sight, muscæ volitantes, or hemiopia. The ophthalmoscope, moreover, very frequently reveals a more or less well-marked inflammation of *both* optic discs (*optic neuritis*). The *hearing* is often affected, and there may be deafness in one or both ears, with buzzing noises. Disorders of sensibility characterised by anæsthesia or hyperæsthesia may be experienced in various parts of the body, especially in the face and extremities. 5. *Motor disturbances.* Paralysis may be entirely absent. When present it may take the form of *hemiplegia*, which under these circumstances usually *comes on gradually.* It is sometimes associated with a defect in one or more of the conjugate movements of the eyes. Partial recovery alternating with relapse is not uncommon. Sometimes the paralysis is limited to a single limb, or to the face. *Local paralyses* are of very common occurrence (whether hemiplegia be present or not) and are due to direct implication of the affected nerves or their nuclei of origin. Thus there may be paralysis of the sixth nerve, causing internal squint; or paralysis of the third nerve, causing external squint with ptosis and dilatation of the pupil; or of the facial nerve; and so on. *Convulsions* are also common and often constitute the first sign of mischief. They may be general, but are more frequently *limited to a single part* or *to one side of the body.* 6. *Mental disturbance* is generally a late symptom and is characterised by gradually advancing imbecility. *Death* usually occurs from convulsions or coma, or a combination of both.

CEREBRO-SPINAL SCLEROSIS.

Sclerosis is characterised by atrophy and degeneration of the proper nerve-elements, associated with a secondary overgrowth of the neuroglia. It occurs most frequently between twenty and thirty years of age, and is sometimes traceable to cold or alcoholic abuse.

Anatomical Characters.—This form of sclerosis consists of small, rounded, dense, and well-defined nodules of a grayish translucent appearance, scattered here and there in the *white* substance of the brain or cord or both. The gray matter is rarely affected. Microscopic examination reveals a fibro-nuclear overgrowth of the neuroglia, with loss of the medullary substance of the nerve-fibres. It is probable that the axis-cylinders of the latter persist to a great extent.

Symptoms.—From the above remarks, it is obvious that the symptoms will vary in different cases according to the distribution of the sclerotic nodules. In a typical case the motor tract both of the brain and spinal cord is mainly affected, and the following description will therefore be based on that fact.

As a rule, the first indication of mischief consists in the gradual onset of muscular weakness and trembling. The weakness generally begins in one of the lower limbs, and shortly afterwards extends to the other. As time goes on, it gets worse and worse and amounts to positive paralysis, though temporary remissions in its progress are not infrequent. There is no loss of power over the rectum or bladder, and it is not accompanied with any disturbance of sensibility. The tremor occurs only on attempting any voluntary movement of the affected part, and ceases entirely when the muscles are at rest, and during sleep. Later on, similar paralysis and tremor extend to the muscles of the upper limbs, trunk, head, face, tongue, and lips. There is no loss of muscular sense, neither is the unsteadiness increased by closure of the eyes. There is some increase in the reflex irritability; thus the knee-jerk is exaggerated, and ankle-clonus is readily obtained. The nutrition and electric excitability of the muscles are unaffected. The speech is slow, syllabic, and hesitating; this is partly due to a mental defect, and partly to a difficulty of articulation. Oscillation of the eyeballs (*nystagmus*) is a frequent symptom. The mind at this stage is usually fairly clear, but there may be some amount of headache and vertigo.

As the case proceeds, attacks of *tonic spasm* are apt to occur in the paralysed muscles, which tend to become more and more frequent, till at last the limbs become permanently rigid. Also as the brain becomes more involved, the mind begins to suffer, the memory fails, the will becomes weak, and the emotions are readily excited. Ultimately the patient lapses into a condition of paralytic dementia. The progress of the disease is tedious, the duration sometimes extending to eight or ten years, and death ultimately occurs from asthenia, or some complication.

If the lesion extends to the posterior columns, or to the anterior cornua of the cord, ataxic or atrophic symptoms respectively may complicate the case.

HYDROCEPHALUS.

The term hydrocephalus or "water on the brain" may be applied to any excess of fluid in or beneath the arachnoid, or within the ventricles. It occurs sometimes in connection with shrunken brains met with in old and demented persons; or again with inflammatory affections of the brain and membranes, notably tubercular meningitis, which is sometimes termed *acute* hydrocephalus. In the present chapter, the term is limited to chronic non-inflammatory effusions into the ventricles, which are met with for the most part, but not quite invariably, in young children, in whom the affection is usually congenital. Sometimes, however, it is acquired; and then it appears to be most commonly associated with rickets. The pathology of these cases is very obscure. Occasionally the condition occurs in adults, as the result of pressure on and occlusion of the *venæ Galeni*.

Anatomical Characters.—The ventricles are distended with a clear limpid serum, which varies in quantity from a few ounces to several pints. The lining membrane is often rough and somewhat granular. If, as usually happens, the cranial sutures are not yet united, the effect on the surrounding parts is very striking. The convolutions are effaced and flattened against the skull, which meanwhile greatly enlarges in its upper part; the fontanelles and sutures greatly widen, and the bones are thinned.

The symptoms may be arranged in three groups:—1. *The appearance of the head.*—This becomes enormously enlarged, and is only supported with difficulty; the forehead is broad and projecting; and the orbital plates are depressed so that the eyes are prominent, and look downwards. The fontanelles and sutures are wide, and may fluctuate on palpation; and the superficial veins are very distinct. The face appears very small by contrast, and wears a curious careworn expression. 2. *Secondary nervous symptoms.*—Convulsions often constitute the earliest symptom, and are apt to recur from time to time. The child is fretful and dull, and the mind gradually fails, culminating perhaps in imbecility. The gait is tottering, and the limbs are weak and subject to convulsive twitching. Sight and hearing may be impaired or lost. 3. *Failure of nutrition.*—The body wastes though food is often taken greedily, and the bowels are generally constipated.

The duration of the disease varies much. Death more often occurs during the first or second year from convulsions, coma, or some complication; but it is occasionally deferred till much later. When hydrocephalus comes on subsequently to the union of the cranial bones, the symptoms are very obscure, and a correct diagnosis is a matter of great difficulty.

GENERAL PARALYSIS OF THE INSANE.

Although the plan of this work does not include mental diseases, it seems desirable to shortly refer to general paralysis, as dependent upon organic disease of the brain. It is characterised by progressive paralysis in association with peculiar mental perversion. It is most frequent in men between 30 and 50 years of age. Sexual excesses, alcoholism, syphilis, and nervous overstrain rank as predisposing causes.

The order in which the symptoms develop varies in different cases, but as a rule the disease begins with mental and moral disturbance, such as irritability, restlessness, tendency to extravagance, failure of memory, and carelessness of appearance and manner. After a variable interval, signs of muscular weakness appear—tremor of the lips, tongue or hands on voluntary movement. The pupils at this stage are often unequal. From this point onwards, the history of the case is one of continued mental and physical deterioration. On the one hand, the patient becomes more and more incapable of mental exertion, and delusions, usually of an extravagant and optimistic kind, develop. Maniacal outbursts are of frequent occurrence, and the patient finally drifts into complete fatuity. On the other hand, muscular weakness extends over the whole system, the faculty of articulation is lost, general paralysis supervenes, bed-sores develop, and in the last stage the patient lies perfectly helpless. Convulsive attacks are common. Remarkable remissions in this downward course are occasionally met with. The affection is almost invariably fatal, after a duration rarely extending beyond two years.

The morbid appearances most frequently observed are thickening of the cerebral membranes with degeneration of the cortical substance.

The treatment is eminently unsatisfactory; and little can be done beyond strict attention to the diet, careful hygiene, and prevention of bed-sores.

SUB-SECTION II.

DISEASES OF THE SPINAL CORD.

SPINAL MENINGITIS.

FOR reasons set forth in the chapter on cerebral meningitis it is convenient to distinguish between spinal pachymeningitis, arachnitis, and lepto-meningitis respectively; and like that disease, the first two varieties are exclusively caused by surgical disease or injury, and so need not be further alluded to here. Lepto-meningitis often arises in a similar way, but in this case—as in the cerebral form, all the membranes are involved. Simple or idiopathic and tubercular spinal meningitis are examples of pure lepto-meningitis. The former occurs in connection with certain acute diseases or from exposure to cold and wet, and may be either acute or chronic. The tubercular form is always acute. Spinal meningitis may exist alone, but it is frequently associated with cerebral meningitis.

ANATOMICAL CHARACTERS.—The *acute* form is characterised by active congestion of the pia-mater, and effusion of turbid, flocculent, or semi-purulent products beneath the arachnoid. These changes are of course associated with tubercles in the tubercular variety. In *chronic* meningitis, the membranes are thickened and opaque, and more or less adherent to each other and adjacent parts, with or without effusion. Acute inflammation generally involves the whole length of the meninges, while the chronic form is frequently limited to some one part. Corresponding changes also extend to the cord.

The SYMPTOMS of simple and tubercular meningitis are alike on the whole, and are due at first to direct irritation, and afterwards to loss of function of the surface of the cord together with the motor and sensory roots of the spinal nerves. *Acute meningitis* begins with chilliness, followed by fever. At the same time there is *intense pain in the back*, which is aggravated much more by movement than by pressure, and is accompanied with sharp radiating pains in the limbs. The sensibility of the skin is also painfully exaggerated. Another prominent feature is tonic muscular spasm, affecting the trunk and limbs, and causing various contractions of those parts. The above signs of irritation gradually give place to those of depression. Thus muscular spasm is replaced by paralysis, and hyperæsthesia by anæsthesia. Reflex excitability which was previously increased becomes diminished. As a rule the paralysis is not complete, and the bladder and rectum are not prominently affected. It will, however, be seen that the symptoms of meningitis are really those of superficial myelitis, and their severity depends upon the extent to which the latter proceeds. When associated with cerebral meningitis (as always happens in the tubercular form), the signs and symptoms of this condition are superadded.

Recovery is quite exceptional, and in the course of two or three weeks, death occurs from asphyxia (by paralysis of the respiratory muscles), or asthenia. Sometimes the case passes into a chronic form.

The SYMPTOMS of *chronic meningitis* are not very unlike those of the acute form, but the onset is more gradual, there is no fever, and the progress is slower. The *pain* in the limbs is frequently much more marked than that in the back, and resembles "rheumatic pains." It is commonly associated with more or less *tonic or clonic spasm*, and exaggerated sensibility. Incomplete paralysis gradually develops, but anæsthesia is rare. *Marked fluctuation in the intensity of the symptoms from time to time* constitutes an important feature of chronic meningitis. Death is the usual termination, and may be preceded by bed-sores, purulent cystitis, or pulmonary complications.

SPINAL HÆMORRHAGE.

HÆMORRHAGE either in the substance of the cord, or the membranes, is almost always the result of injury. Occasionally, however, it arises from the rupture of an aneurism.

Symptoms.—Hæmorrhage into the substance of the cord is followed by the *sudden* suspension of motion and sensation in the lower half of the body, together with paralysis of the bladder and rectum. Unless the lesion be so high up as to cause asphyxia by paralysis of the respiratory muscles, death may be deferred for years, ultimately occurring from bed-sores, cystitis, or some other complication.

In meningeal hæmorrhage such complete functional annihilation develops more gradually, and is due to compression of the cord by the effused blood. The earlier symptoms consist of pain in the back with tonic and clonic spasms indicative of severe spinal irritation. Under all circumstances the diagnosis rests upon the history of the case, and the *sudden* onset of symptoms.

ACUTE MYELITIS.

Acute Myelitis or inflammation of the *substance* of the spinal cord may be caused by injury, spinal caries, meningitis, or exposure to extreme heat or cold, &c.

The *anatomical changes* consist of active congestion, followed by softening which is frequently combined with hæmorrhagic extravasation. Suppuration is very rare. When secondary to meningitis the superficial white substance is first affected, and the more deeply-seated gray substance is not involved till later on. Idiopathic inflammation, on the other hand, generally begins in the central gray substance. In either case the tendency is to implicate the whole thickness of the cord together with its membranes in a variable part of its length.

Symptoms.—Acute myelitis generally begins with chilliness, followed by a considerable rise of temperature and general febrile disturbance. *Pain in the back* (not so intense as in meningitis) is an early and prominent symptom. It is increased by sharp percussion, and often by the application of a hot or cold sponge to the part, but not so much by movement. This is usually accompanied with a sense of painful constriction, extending round the body like a girdle, at the level of the upper limit of inflammation. Anæsthesia and paralysis occur early, and rapidly become complete. They involve all the parts and muscles supplied by nerves issuing from the affected segment of the cord and that which lies below the lesion. The bladder and rectum are early involved, resulting in incontinence of urine and fæces. The temperature of the paralysed parts is always depressed. Reflex and electric irritability may be exaggerated just at the outset, but soon become impaired and finally lost. Similarly, the affected muscles are flaccid, and rapidly atrophy; visceral lesions occur, of which cystitis and acute nephritis are the most common, and acute bed-sores are of frequent occurrence. In those cases of myelitis which are secondary to meningitis, as so frequently happens, the early stages will of course be characterised by the symptoms peculiar to the latter affection; and under any circumstances there are generally indications of meningeal irritation at the upper level of the lesion. Acute myelitis is generally fatal and death usually takes place in the course of one to four weeks from exhaustion or some complication; but sometimes a partial or complete recovery is made.

Chronic myelitis usually gives rise to indurative changes in the cord, which are now described under the name of "sclerosis," a general account of which is given in the next chapter. But there is one variety, known as "transverse myelitis," calling for brief description here, in which the whole thickness of the cord for a variable length is sometimes softened instead of being sclerosed, especially when it has developed from, or ends with an acute attack. It may be caused by concussion or gradual compression of the cord, or by exposure to cold and damp, over-exertion, chronic alcoholism, or syphilis. It is most common during youth and middle life, and in the male sex.

The *symptoms* in some respects resemble those of primary lateral sclerosis. There is the same "spastic paralysis" of the muscles supplied by nerves from the cord below the level of the lesion, but the paraplegia is more complete and rapid in its development, and it has little tendency to extend its upper level. Moreover, we meet with anæsthesia and abnormal sensations in the lower parts of the body (though pain is rare), also with bladder-weakness and bed-sores. The general health is at first but little impaired, but recovery is rare; and after a slow chronic course, often marked by intervals of considerable improvement, death occurs from exhaustion or some complication.

SPINAL SCLEROSIS.

SCLEROSIS is a chronic process which primarily consists of atrophy of the proper nerve-elements, accompanied with overgrowth and induration of the neuroglia. It appears in the spinal cord in three forms:—(1) *Multiple sclerosis,* in which the lesions are several in number, and isolated from each other; (2) *cortical sclerosis,* in which the lesion is confined to the cortical substance of the cord; and (3) *funicular sclerosis,* in which one or more of the white columns of the cord are involved for a part or the whole of their length. Multiple sclerosis has been already described under the head of cerebro-spinal sclerosis; and since cortical sclerosis is always associated with chronic meningitis, the symptoms are closely analogous to those of the latter affection. It now remains to describe funicular sclerosis under two heads, according as the lesion is seated in the lateral, or in the posterior columns of the cord.

The *causes* of sclerosis are often obscure, but middle age, hereditary taint, gout, syphilis, alcoholic or sexual excesses, are among those which appear to exert most influence. In most cases it appears to be secondary to inflammatory affections such as meningitis, spinal curvature, spinal tumours, &c.

I. Lateral Spinal Sclerosis.

This affection may be described under three heads:—(1) primary; (2) amyotrophic; and (3) secondary lateral sclerosis.

Primary lateral sclerosis.—The first symptom usually consists of progressive weakness of the legs, which at an early period becomes associated with spasmodic muscular contractions, gradually developing into more or less permanent tonic rigidity of these parts, thus rendering walking difficult or impossible. On attempting to walk, the gait is peculiar and characteristic, the legs are kept close together, with a curious tendency to step on tip-toe, and a bending of the body as if about to fall forward. The difficulty of walking is out of all proportion to the amount of paralysis. Ankle-clonus can be readily elicited, and the knee-jerk is greatly exaggerated. The bladder and rectum are not materially affected, though there is often obstinate constipation. So long as the lesion is limited to the anterior lateral columns there is no disturbance of sensibility, and the nutrition and electric behaviour of the muscles remain normal. The legs are first affected—generally one before the other—but later on the arms may also become involved. In the last stages of the disease, the rigidity passes off, the paralysis increases, the muscles atrophy, and the knee-jerk is diminished, probably indicating the extension of mischief into the anterior cornua. The course is very slow and extends over many years as a rule. Death usually takes place from some intercurrent disease. The disease often goes by the name of "spastic paralysis," and the peculiar gait is called the "spastic gait." It is most common from thirty to fifty years of age.

Amyotrophic lateral sclerosis.—This rare disorder is characterised pathologically by primary sclerosis of the lateral columns of the cord and degenerative atrophy of the anterior gray cornua. The change is most marked in the cervical enlargement, and affects both sides of the cord symmetrically. Its etiology is very obscure; but it occurs more often amongst women from twenty-five to fifty years of age.

In the first instance, the muscles of the arm become weak, atrophied, painful on pressure, and rigid from tonic contraction. The limbs are flexed, the wrists are pronated and the fingers closed on the palms of the hand. There is no anæsthesia, and no paralysis of the rectum or bladder. The legs may be subsequently affected. Sooner or later the signs of bulbar paralysis supervene, to which the patient finally succumbs.

Secondary lateral sclerosis.—The circumstances under which this condition occur have already been described in a previous chapter. Rigidity of the muscles supplied by nerves in connection with the affected tract, tonic contractions, and increased tendon-reflex are the chief outward results of this lesion.

2. Posterior Spinal Sclerosis—Locomotor Ataxy—Tabes Dorsalis.

The sclerosis here occupies the posterior columns of the cord; but though in many cases each column is affected in its whole thickness, the essential lesion on which the characteristic symptoms depend affects that portion of the so-called posterior root-zone next to and on the inner side of the posterior gray cornua. The change is usually most marked in the lumbar enlargement, but it is frequently met with in the upper part of the cord. Analogous lesions may also occur in the brain, notably in the restiform bodies, inferior cerebellar peduncles, optic thalami, and in connection with the optic, auditory, third, and sixth nerves.

Locomotor ataxy is sometimes hereditary, and is certainly more common in those with a family history of nervous diseases, than in others. Syphilis and intemperance appear to be especially frequent predisposing causes. It is most common in men of middle age.

Symptoms.—In the early or so-called *pre-ataxic stage* the symptoms vary very much in different cases; so much so that a precise consecutive account for general application is impossible. One of the commonest and earliest symptoms consists of sharp "*lightning pains*" down the limbs, often associated with dull pains in the back, a feeling of constriction round the body, numbness, and formication. Paroxysms of gastralgia accompanied with vomiting (*gastric crises*) also occur in certain cases. Analgesia (loss of painful impression) and to a less extent anæsthesia (loss of touch) constitute common disturbances of sensation; and are apt to exhibit a patchy distribution, which may vary from time to time. Muscular sense is also impaired in the affected parts, so that the patient is unaware of the position of his limbs, unless assisted by ocular observation, and he is unable to appreciate differences in weights attached to his limbs. Among other early symptoms, the following deserve close attention :—(1) Abolition of the knee-jerk. This sign is of the very greatest diagnostic value, for it is rarely if ever absent. (2) Visual disturbances. The pupil is generally contracted, and in many cases is insensible to the stimulus of light, while it continues to contract in the accommodation of the eye for near objects (*Argyll-Robertson phenomenon*). The ocular nerves—especially the third and the sixth—are often paralysed, giving rise to squint. This symptom is sometimes of only transient duration. Atrophy of the optic disc is also a common ophthalmoscopic sign. (3) Disturbances of hearing, taste, and smell may be also observed. (4) Sexual desires may be impaired or lost; or (less frequently) we may meet with persistent erections, with increased capacity for frequently repeated intercourse. (5) The functions of the bladder and rectum are very frequently disturbed. Just at first there may be some irritability of the bladder, but later on there is a difficulty in emptying the organ. Constipation is the rule; and not infrequently shooting pains, and marked discomfort in the rectum are complained of.

Concurrently with some or all of the above symptoms there is *gradually increasing muscular inco-ordination.* In the first instance it does not amount to true ataxy, but the gait is uncertain, and the patient is apt to sway about in the dark, or if the eyes be closed. There is *no loss of muscular power;* and until the close, the nutrition and electrical reaction of the muscles remain intact. In course of time true ataxy develops (*ataxic stage*), and the patient is unable to stand with his feet together and his eyes shut, while the gait is characteristic. The act of walking requires the closest watchfulness on the part of the patient, the leg which is in movement is thrown irregularly forward with unnecessary violence, and is brought to the ground—heel first—with unnecessary force, until at last his movements become so uncertain and tumultuous, that walking is impossible. But with all this, so long as he can walk at all, he may not become fatigued very readily.

Curious trophic lesions may also occur, of which the following are the chief:—temporary œdema, herpes, perforating ulceration of the foot, special brittleness of the bones leading to so-called "spontaneous fractures," and certain joint changes closely similar to if not identical with those of rheumatoid arthritis.

Locomotor ataxy, though occasionally acute, generally runs an extremely tedious course, extending over many years. A single symptom may exist alone for a long time, but sooner or later others appear, and gradually tend to become worse and worse. In the later stages, as above noted, the patient becomes unable to walk, gradually losing the power of performing separate movements. The muscles become soft and flabby, and undergo atrophy. Anæsthesia of the affected limbs may become more or less complete, bed-sores may develop, the urine and fæces may dribble away, and the patient may finally die from sheer exhaustion. More often, however, death occurs from some intercurrent trouble, such as renal disease or general paralysis of the insane. The terminal symptoms just alluded to are described as the *paralytic stage* of the disease, by some authors.

TUMOURS OF THE CORD.

New growths or tumours, similar to those which are met with in the brain, may also occur in the spinal cord.

The symptoms to which a tumour gives rise, will necessarily vary according to its seat, size, and rapidity of growth; in one case resembling those of meningitis, and in another those of some form of chronic myelitis, as the membranes or the substance of the cord respectively happen to be involved. Thus a certain diagnosis is only possible in exceptional cases.

GLOSSO-LABIO-PHARYNGEAL PALSY—BULBAR PARALYSIS.

THIS disease as its name implies, is characterised by a paralysis of the tongue, lips, soft palate and pharynx. It is rare under forty years of age Though it occurs as a separate affection, it sometimes forms a part of other nervous disorders, *e.g.*, multiple cerebro-spinal sclerosis, and progressive muscular atrophy. Its causes are very obscure. *Pathologically*, it depends upon pigmentary degeneration and atrophy with some secondary sclerosis of the nuclei of the hypoglossal, facial, spinal, accessory, and vagus nerves in the medulla oblongata.

SYMPTOMS.—The onset is gradual. The first symptom usually consists of a difficulty in articulation due to a want of precision in the movements of the tip of the tongue. The *paralysis of the tongue* increases till at last it can be no longer protruded, and lies motionless, or nearly so, in the mouth. Meanwhile the paralysis slowly involves other parts. The *soft palate* hangs loosely in the throat, and gives a nasal twang to the voice. The *constrictors of the pharynx* fail to grasp the food properly, and cause a difficulty in swallowing. The muscles of the larynx also become weakened. Curiously enough while the orbicularis oris becomes paralysed, so that the lips cannot be kept together, the other facial muscles are not affected. As the symptoms get worse, the condition of the patient is pitiable indeed. Articulate speech is impossible, though phonation is not entirely lost; the saliva cannot be properly swallowed, and accumulating, dribbles from the open mouth; food collects in the sides of the mouth, can only be swallowed with great difficulty, and is apt (especially if liquid), to enter the larynx or to return through the nose. As a rule the nutrition of the affected muscles, as well as their electrical behaviour, remains normal; but sometimes they undergo atrophy, and exhibit the reaction of degeneration (Erb). The *general health*, though good in the early stages, gradually suffers from insufficient nutrition and imperfect respiration.

It is always fatal, the average duration being about two years. Death occurs either from starvation, asphyxia, syncope, or some intercurrent disease, as bronchitis.

ANTERIOR POLIO MYELITIS.

This somewhat unwieldly term literally means inflammation of the anterior gray substance of the cord, and is now generally used to denote certain pretty-well-defined lesions of the large nerve-cells of the anterior cornua followed by paralysis and muscular atrophy. The acute form includes the disease usually described as "infantile paralysis," and acute spinal paralysis of adults; while the chronic form is better known as "progressive muscular atrophy."

Acute Anterior Polio-myelitis.

Acute anterior polio-myelitis is an acute form of paralysis chiefly met with in young children, especially during the second year of life; but adults are occasionally affected. It may sometimes be traced to exposure to cold or wet, difficult dentition, or to some acute febrile disorder; but more often its causes are obscure.

The lesion on which this disease depends, is an acute inflammation of the anterior cornua of the cord, which results in the atrophy and destruction of the ganglion-cells, with an infiltration of small round cells, and granular corpuscles. This is followed, at a later stage, by sclerosis of the cornua, the adjacent parts of the antero-lateral columns, and of the anterior nerve-roots.

SYMPTOMS.—The onset is *sudden*, and marked by *fever* which may be considerable and continue for some days, or be so slight as not to attract attention. The *paralysis* develops quickly, and may be limited to a single group of muscles, or to one limb; or it may affect both arms or legs, or all four limbs at once. From the first the muscles are flaccid, and both reflex and faradic excitability quickly become impaired and lost. Galvanic irritability is at first increased. The bladder and rectum are not involved; there is no pain; and cutaneous sensibility is not affected. Having arrived at this stage, the paralysis may gradually pass off with complete recovery. This favourable termination is more common in adults than in young children. More frequently in the latter, special groups of muscles, or the whole of one or more limbs, remain permanently paralysed. In this case *the affected muscles rapidly atrophy;* the temperature of the part falls, with more or less arrest of its development; and permanent deformities may arise from the unrestrained action of non-paralysed muscles. The *general health* is not impaired. The disease is never fatal, but its duration is extremely variable.

Chronic Anterior Polio-Myelitis—Progressive Muscular Atrophy.

This affection is most common in males between twenty-five and thirty-five years of age, and appears to be frequently hereditary. Sometimes the exciting cause is excessive exertion, sometimes exposure to cold and wet, but more often it is quite obscure.

The lesions found in the cord essentially consist of sclerosis of some part or parts of the anterior cornua. The motor nerves arising from these parts, and the muscles in connection with them, rapidly atrophy and degenerate.

Symptoms.—The onset is insidious. Attention is first attracted by weakness (sometimes accompanied with pain) in the affected muscles, which are then found to be somewhat atrophied. This atrophy and weakness slowly increase. The change usually begins in the right hand, the thenar and hypothenar eminences of which are flattened, and the outline of the metacarpal bones becomes more distinct from atrophy of the interossei and adjacent muscles. In other cases the muscles of the forearm or shoulder, or—more rarely—analogous muscular groups in the leg are first attacked, the atrophy of which leads to striking changes in the appearance and outline of the part. Wherever the disease begins, it first extends to corresponding muscles on the opposite side of the body, and then in like manner to the trunk and all the limbs. It should be clearly understood that *the paralysis results exclusively from muscular atrophy, and is in direct proportion to its extent. Fibrillary oscillations* are of common occurrence in the atrophied muscles, and when absent may usually be evoked on irritating them by tapping, pinching, &c. Since all the muscles of a limb are not involved simultaneously or to an equal extent, *deformities* occur from their unequal tension. The *electric contractility* is only diminished in proportion to the amount of atrophy, and the *temperature* of the affected part is always lowered.

The course is always slow, and advances by fits and starts. Its duration is very indefinite. Death ultimately takes place from the respiratory muscles becoming involved, from exhaustion, or from some intercurrent affection.

PSEUDO-HYPERTROPHIC PARALYSIS.

Although no precise pathological position has as yet been assigned to this disease, it will be convenient to consider it in this place. It is characterised by progressive weakness of certain muscles, associated with their enlargement. It has rarely been observed except among children, and is much more frequent among boys. As regards its pathology little is definitely known, but there is evidently a pretty close relationship with "progressive muscular atrophy," and perhaps it is essentially the same.

The muscular enlargement, as the name implies, is not a true muscular hypertrophy, but is due to an excessive overgrowth of the connective tissue between the fibres, which ultimately atrophy and degenerate. Certain atrophic changes of the anterior cornua and motor roots of the spinal nerves have also been observed in a few cases.

Symptoms.—The earliest symptoms consist of weakness of the legs, and a peculiar, straddling, waddling gait. The spine is arched backwards and the belly is thrust forward. Soon the calves of the legs are found to be unduly large and prominent; and subsequently the thighs and gluteal muscles. As the enlargement increases, the paralysis becomes more marked, until at last both walking and standing are impossible. Though usually confined to the parts mentioned, the muscles of the trunk and arms are sometimes involved. Later on, the enlargement of the muscles ceases, and they may even diminish in size. The course of the disease is slow, the average duration being about five or six years. Death takes place from exhaustion, implication of the respiratory muscles, or some intercurrent disease.

PARALYSIS AGITANS.

This is an affection of advanced life, and is mainly characterised by trembling. Little or nothing is definitely known respecting its causation or morbid anatomy.

Symptoms.—The onset is almost always insidious, and is first manifested by *trembling*, which usually begins in the hand or foot of one side. It is present whether voluntary movements are performed or not, but is increased by mental excitement or physical exertion. It ceases during sleep. It may be confined to the part in which it began for months; but sooner or later it extends to the rest of the limb, then usually to the other limb of the same side, and then ultimately to the limbs of the opposite side. As a rule, it is not until the tremor has lasted a long time, that weakness of the affected muscles is observed, but when once it has begun, it slowly becomes more marked.

The *muscles are apt to become more or less rigid* as the disease proceeds. The *speech* is not characteristically altered, but may be a little trembling. The *gait* also is peculiar; the patient appears to be losing his balance and running forward to save himself, scarcely lifting his feet from the ground (*festination*). The muscles of the face are not involved; there is no nystagmus; there is no alteration of sensibility; and there are no head-symptoms. Death does not usually occur till after some years, and is then caused by exhaustion, or some intercurrent disorder.

GENERAL TREATMENT OF CENTRAL NERVOUS DISORDERS.

In *acute inflammation of the brain and membranes*, our chief therapeutic resources are of an indirect kind. The patient's room should be cool, well ventilated, and quiet; the hair should be cut short, or (in severe cases) shaved to the scalp, followed by the application of cold in the form of an ice-cap or cold-water coil, or by the application of a few leeches to the temples or behind the ears if there be much pain and irritability. The bowels should be actively opened from time to time by a brisk aperient, such as calomel and jalap—gr. x. of each for an adult, and $\frac{1}{3}$ to $\frac{1}{2}$ that dose for a child. As general calmatives, in addition to the cold applications to the head already mentioned, the most useful are—the tepid wet sheet pack, mustard pediluvia, or the wrapping round the feet and legs with cloths wrung out in strong mustard and water, bromide of potassium, chloral hydrate, and morphia. The latter should be used cautiously, but the addition of a small quantity to a bromide or chloral draught at bed-time is often advantageous, especially if there is much pain. When opiates are contra-indicated as with young children, tincture of henbane may be substituted. Where there is active congestion full doses of ergot given periodically (with a saline effervescent if there be much fever) or hypodermic injections of ergotine are worthy of trial. The diet should be bland, plain, and nutritious; and if there be any difficulty about feeding by the mouth, nutrient enemata must be persevered with. Alcohol is not necessary as a rule; but in the later stages where there is marked prostration it may be needed.

Acute inflammation of the cord and its membranes requires analogous treatment. Dry cupping alongside the spine affords a further means of some value, in addition to those already mentioned, for the purpose of reducing local congestion. In all acute central nervous affections, the bladder should be periodically examined with care, especially in affections of the cord, and if there be retention, the urine should at once be drawn off. If the bladder be inflamed, it should be frequently washed out with some anti-putrefactive solution, such as boracic acid (sat. sol.) or hydrarg. bichlor. (1 in 3000).

The treatment of *chronic cerebral and spinal disease* is far from satisfactory. Physiological rest of the nervous system should be aimed at; and the general health must be supported by good food, fresh air, and the use of tonics and cod-liver oil. In many cases the internal free administration of iodide of potassium and mercurials is most beneficial, especially in those of a syphilitic nature. Phosphorus, phosphide of zinc, Donovan's solution, and nitrate or oxide of silver are also serviceable nervine tonics in chronic degenerative diseases of central nervous system.

The present is also a convenient place for referring to the leading principles of the treatment of the more important symptomatic states referable to nervous disturbance. *Headache.* Careful search for likely causes must be made, *e.g.*, carious teeth, visceral disturbance, exhaustion, gastro-intestinal trouble (especially constipation), rheumatism, gout, syphilis, and organic cerebral disease; and endeavours should be made for their correction or removal. Too much stress cannot be laid upon the importance of good hygiene in all classes of cases. Since the toxic effects of imperfectly metabolised products are not only a common primary cause of headache, but a frequent factor in combination with other causes, a light simple dietary, and moderately free elimination from the bowels and kidneys, are generally beneficial. Active purgation, however, should be avoided in the weak, feeble, or aged. The following hints may be of service. In anæmia—ferruginous tonics, arsenic, strychnia, phosphorus, cannabis indica, and alcohol; in congestion—bromide of potassium, ergot, cold applications to the head, and moderate purgation; in dyspepsia—alkalies before meals, mineral acids after meals, bismuth, nux vomica, vegetable bitters, and alterative aperients; in nervous exhaustion—rest, tonics, phosphorus, valerianate of zinc, and (if sleepless) bromide of potassium, chloral, or morphia at night under strict supervision; in syphilis—iodide of potassium; and in headaches from organic disease—bromides, gelsemium, cold applications to the head, sinapisms to the back of the neck, or leeches behind the ears.

Vertigo. Common causes like anæmia, gastro-intestinal derangement, ocular troubles, abuse of alcohol, tea, coffee, or tobacco, should be enquired for and removed. In aural vertigo, full doses of quinine with hydrobromic acid, bromide of potassium, or a hypodermic injection of morphia will often relieve an attack at the time, but they do not prevent its recurrence, or interrupt progressive deafness.

Insomnia. The chief pathological causes of sleeplessness are over-exhaustion, fever, congestive conditions of the brain, gouty dyspepsia, and certain toxic agents such as alcohol, tea, coffee, and tobacco. Insomnia from exhaustion often demands prolonged rest and change of air and scene. Mustard pediluvia, or a hot full bath at bed-time are of service. Such patients often sleep fairly well for the first part of the night, but wake up in the early hours, feeling low and depressed, and get no more real sleep till morning. In these cases, not only should some light nourishment and a little stimulant be taken before retiring to bed, but it should be repeated on waking—once or oftener as required. As to medicines, opiates and the bromides are objectionable, and chloral or paraldehyde are the best hypnotics. A course of nervine tonics is also most beneficial. Insomnia during the early stage of fevers is best combated with cold to the head, cold spongings, and tepid wet sheet packing; and, if accompanied with much excitement—bromides or chloral. During the later stages, a frequent supply of nutritious food, discreet recourse to alcoholic stimulants, and careful attention to the excretory functions are specially indicated. The principles of the treatment of congestive headaches are similar to those already given for active cerebral congestion. Sedative drugs may be necessary, but opiates should only be given with caution. The treatment of dyspeptic and toxæmic insomnia calls for no special remark in this place.

The treatment of *delirium* will vary according to its cause. When it is due to cerebral inflammation, the measures already mentioned must be adopted; when it results from exhaustion and the skin is cool with a feeble pulse, good food and stimulants are called for, and not infrequently an opiate is of great service unless there be coma, renal disease, or pulmonary congestion. In the "typhoid state" alcohol, nourishing food, and tepid sheet-packs, with careful attention to the excretions, are necessary. In delirium from high fever the cold bath or cold sheet-packs are of most benefit; and lastly, if traceable to reflex irritation, the cause of the latter should be carefully investigated and removed if possible, and opium, chloral, or bromide of potassium should be given internally.

The treatment of *coma* also varies according to its cause. In apoplectic coma the patient should be placed in the recumbent posture with the head high, allowed plenty of fresh air, and the clothing loosened. Venæsection may be of benefit if the circulation be vigorous and the breathing free, but under other circumstances it is more harmful than otherwise. The bowels should be freely opened, by placing a drop or so of croton oil in a little butter on the back of the tongue. If the coma be associated with renal disease, it may be uræmic, and then calls for the treatment already described under that head. Coma from opium poisoning, &c., requires prompt use of the stomach-pump, followed by the administration of strong coffee, and constant flipping and movement in order to keep the patient roused.

In chronic *paralytic* conditions from central nervous disease local treatment is very useful, such as counter-irritation to the spine, systematic rubbing of the muscles (*massage*), and the various forms of electrisation.

SUB-SECTION III.

DISEASES OF THE NERVES.

NEURITIS.

NEURITIS, or inflammation of nerves, may be set up by local injury, pressure of bones in fractures and dislocations, exposure to cold and damp, adjacent inflammation, and by certain general conditions such as syphilis, gout, rheumatism, chronic alcoholism and lead-poisoning, also as a sequel of some specific fevers. Usually only one nerve-trunk is affected, but in some cases—especially when dependent on chronic alcoholism—many are affected at the same time (*multiple neuritis*). Neuritis may be *acute* or *chronic*.

The inflammation may chiefly affect the interstitial connective-tissue, causing thickening and induration of the nerve, followed by atrophy and degeneration of the nerve-fibres; while in other cases, the process begins in and chiefly affects the nerve-fibres.

In *acute neuritis* there is severe pain and tenderness along the course of the nerve, extending into the parts supplied by it. It is increased by movement or pressure. Sensation is perverted from the first, and ultimately it is diminished or lost. The muscles supplied by the affected nerve may exhibit fibrillary twitching or painful cramps, and after a time they become weak, flabby, and atrophied, showing the reaction of degeneration. In *chronic neuritis* we also meet with trophic changes, *e.g.*, cutaneous eruptions, a peculiar glossy condition of the skin, irregularities of nails, and atrophy of hairs. In *alcoholic multiple neuritis* the local condition is much the same, but in addition there is a good deal of constitutional disturbance and fever in the first instance. The limbs are symmetrically attacked, the numbness and paralysis gradually extending from the extremities upwards. The extensors suffer much more than the flexors, and chronic flexures of the limbs may thus occur. The reflexes are lost and the mind is generally more or less enfeebled. Muscular inco-ordination is sometimes observed, and such cases are apt to be confounded with locomotor ataxy.

The course of neuritis is generally tedious; but if properly treated the prognosis is not unfavourable. A certain degree of improvement is the rule, and complete recovery is not uncommon.

The principles of *treatment* are as follows:—The primary cause should be carefully enquired for and removed as far as possible. Locally, soothing applications are indicated during the acute stage; while chronic neuritis demands massage and electrisation. Pain may be relieved by morphia injections. Medicinally, iodide of potassium and mild mercurials are of most service; and in all cases, attention to the general health is of the greatest importance.

OCULAR AND AUDITORY DEFECTS.

Optic neuritis is characterised by swelling and congestion of the optic disc, and more or less blurring of its outline, revealed by ophthalmoscopic examination. Vision is not necessarily impaired. Double optic neuritis is a common result of irritative intra-cranial disease, of which it is valuable confirmatory evidence. It also occurs in chronic Bright's disease, lead-poisoning, and certain disorders of the blood. Unilateral optic neuritis may be caused by local disease in the orbit.

Atrophy of the optic disc is often the sequel of inflammatory mischief, but is sometimes primary, as in locomotor ataxy, for instance. The disc is pale and excavated, its margin is distinct, and the retinal vessels are small.

Hemianopia, or blindness over half of the field of vision, results from destructive lesions of the optic tract on the opposite side, or from a lesion in the occipital lobe of the brain. Injury of the optic commissure generally causes blindness in the outer half of each field of vision (*temporal hemianopia*).

Diplopia, or double vision, results from a want of parallelism in the axes of the eyes, or squint. In convergent squint, each image corresponds to the eye of the same side (*lateral diplopia*); while in divergent squint, the right image corresponds with the left eye, and *vice versâ* (*crossed diplopia*).

Variations in the pupils.—*Myosis*, or persistent contraction of the pupil, may be due to hypermetropia, paralysis of the sympathetic in the neck, photophobia, hæmorrhage into the pons, or certain poisons such as opium. It is common in locomotor ataxy from interference with the cilio-spinal centre in the cervical part of the cord. Dilatation of the pupil, or *mydriasis*, is observed in atrophy of the optic nerve, paralysis of the third nerve, in general debility, anæmia, cerebral compression, and from certain poisons as atropine or cocaine. *Inequality* of the pupils is common in paralysis of the insane, and aneurism of the aortic arch.

Nystagmus is characterised by oscillations of the eyeballs from side to side, affecting both eyes symmetrically. Amongst the more common causes may be mentioned—albinism, cataract, cerebellar tumours, and multiple sclerosis. Miners are also especially liable to it.

Deafness may be due to a defect in the auditory nerve, or to local disease interfering with the conduction of sound through the ear. Nervous deafness may be distinguished from deafness due to faulty conduction, by means of a tuning-fork made to vibrate in contact with the bones of the head. In the former it cannot be heard; while in the latter the sound is intensified.

Tinnitus aurium is characterised by subjective sensations of ringing, rushing, or humming noises in the ear. It may be caused by any local ear-disease, or general disorder which irritates the auditory nerve, and is often a most intractable symptom. It may be associated with deafness and vertigo.

LOCAL PARALYSES.

The signs of paralysis of peripheral nerves are as follows:—

Cranial nerves.—*Third nerve.*—Divergent squint from paralysis of all the muscles of the eye excepting the superior oblique and external rectus; ptosis from paralysis of the levator palpebrarum; and dilatation of the pupil from paralysis of the sphincter of the iris.

Fourth nerve.—By paralysis of the superior oblique muscle of the eye, the pupil is slightly raised above the lower lid, and there is no power of rotating the eye. *Test.*—If the patient be directed to look at an object below the horizontal median plane, he sees double, and the image formed by the affected eye is displaced downwards and inclined on one side.

Fifth nerve.—*Ophthalmic division.*—Loss of sensation over one-half of the forehead, the upper eyelid, conjunctiva, and a part of the mucous membrane and integument of the nose. The pupil also is often contracted. *Superior maxillary division.*—Anæsthesia of the lower eyelid, cheek, side of the nose, and half of the upper lip, gums, and palate. *Inferior maxillary division.*—Anæsthesia of the lateral part of the head, face and ear, half of the lower lip and gums, and half of the tongue, with paralysis of the muscles of mastication. The nutritive lesions of the skin and conjunctiva which so frequently accompany lesions of the fifth nerve or its nucleus, are due rather to *irritation* than paralysis of the nerve.

Sixth nerve.—Convergent squint from paralysis of the external rectus muscle of the eye.

Facial nerve.—Facial or Bell's paralysis may arise from disease affecting the nerve (1) within the cranium;

(2) in its course through the temporal bone; or (3) at its periphery. The first form, which is rare, may be caused by disease of the medulla, or by the pressure of some tumour. The second form is more common, and is generally due to disease of the temporal bone following inflammation of the middle ear. The third form is most common, and usually arises from exposure to cold.

Symptoms.—The following symptoms are common to all three forms.—The facial muscles of the paralysed side are motionless and flaccid; the lines of expression are lost; the eyelid cannot be closed, and remains open; the tears overflow the lower eyelid; the cheek is puffed out on forcible expiration; the mouth is drawn to the opposite side, and food lodges between the teeth and the cheek on the affected side. The paralysis becomes at once apparent on the patient attempting to frown, purse the lips, or show the teeth. Reflex and faradic irritability are quickly lost, but galvanic irritability is at first increased. There is no anæsthesia.

In the *second form*—over and above the symptoms already mentioned—the parts supplied by the chorda tympani and large petrosal branches of the facial nerve are also paralysed. Thus we find disturbances of hearing, flattening of the arch of the palate on one side, with deviation of the uvula to the sound side; and also some impairment of taste, owing to the suppression of the salivary secretion on one side, or to some obscure direct influence of the chorda tympani on the tongue itself.

In the *first form* we may further meet with head symptoms and signs of implication of other cranial nerves.

The facial paralysis which accompanies hemiplegia differs from the above as follows:—(1) The orbicularis palpebrarum is not involved, so that the eye can be closed as usual; (2) it is usually attended with paralysis of the hypoglossal, so that the tongue deviates when protruded; and (3) the tone of the muscles, and their reflex and electric irritability are normal.

Treatment.—When due to disease of the brain or of the temporal bone, treatment must be directed to these conditions. In the third form electricity is most to be relied on. If the muscles respond to faradism that form of current is the best; but if otherwise, slowly interrupted galvanism offers the only chance of doing good.

The effects of paralysis of the *eighth* and *ninth cranial nerves* have already been sufficiently indicated in the description of glosso-labio-pharyngeal paralysis.

Spinal nerves.—It is impossible to describe here in detail the various paralyses of spinal nerves. It must be remembered that each nerve conducts both motor and sensory impulses, and hence paralysis is always associated with anæsthesia in the parts supplied by the nerve below the seat of the lesion; and further when the latter is of an irritative kind, trophic troubles are not unlikely to arise within the area of its distribution. Injury, pressure, and direct implication in disease are common causes of local spinal paralysis. It may perhaps be well to mention that paralysis of the *musculo-spiral nerve*, which is of very frequent occurrence from sleeping on the arm over the back of a chair, closely resembles the paralysis which results from lead-poisoning. The history of the case, the association with the paralysis of more or less anæsthesia, and the inclusion of the supinator longus (which almost always escapes paralysis from lead-poisoning) will, however, suffice to distinguish it.

LOCAL NERVOUS SPASMODIC AFFECTIONS.

"WRITER'S CRAMP," or "SCRIVENER'S PALSY," as the terms imply, is especially common to those who write inordinately, and is characterised by intermittent pain, weakness, and spasm of the muscles of the hand and arm. It is also met with in violinists, tailors, and others whose occupations involve the prolonged use of similar muscles. Its pathology is obscure.

Symptoms.—Its earliest indication is a painful sense of fatigue and weakness, which comes on shortly after commencing to write. Sooner or later this is accompanied with clonic involuntary spasms of the muscles employed, so that the hand-writing is unsteady and scrawly. At first it can be more or less controlled by voluntary effort, but it becomes worse and worse till at last the use of the pen is impossible. Patients then often learn to write with the other hand, but only to find that, after a while, this becomes affected in a similar manner. Different muscles are affected in different cases. Thus it may be confined to the muscles of the hand or of the forearm; or it may extend from one part to another till the whole arm becomes involved. Sensation is not affected, and for all other acts than that which has induced the disorder the muscles behave properly. The prognosis is unfavourable.

"WRY-NECK," or TORTICOLLIS, consists of clonic or tonic spasm of one or more of the following muscles—sterno-mastoid, splenius, and trapezius. Its pathology is equally obscure.

It comes on for the most part insidiously, with uneasiness or pain in the affected side, and jerking of the head from time to time. The direction of the head varies according to the muscles which are involved. At first the spasms are distinctly intermittent, and can be more or less controlled by the will. But they gradually tend to become more permanent and incontrollable, till the head is permanently twisted to one side. Spasm of the facial muscles, or of those of mastication, or of the arm, may sometimes be superadded to the above.

The TREATMENT of these affections is very unsatisfactory. Most improvement may be expected from the employment of electricity. The constant current should be selected, and the poles applied to the nerve of the part, during which the patient should be directed to perform rhythmical movements of the muscles concerned (Poore).

SUB-SECTION IV.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

EPILEPSY.

EPILEPSY is essentially a cerebral disease, though not due to any obvious structural change. It is characterised by a temporary loss of consciousness of sudden onset, and is associated for the most part with a convulsive attack of short duration. It commonly makes its first appearance some time between ten and twenty years of age, and its onset is less frequent as age advances. There is often a distinct history in the family either of epilepsy or of some other nervous disease, insanity, or intemperance. Fright, anxiety, over-work, injuries of the head, and masturbation are common remote causes, but often none can be discovered. As to its pathology, many theories have been advanced from time to time, but it is most probable that an epileptic attack is the direct outcome of sudden explosive action in some part of the cerebral cortex, which is due rather to enfeebled power of restraint, than to any excess of energy.

SYMPTOMS.—A detailed description of all the variations exhibited by epilepsy is here out of the question. Only two typical forms will be considered, the difference between which essentially consists of a difference in severity:—(1) *Minor epilepsy*, or *petit mal;* and (2) *major epilepsy*, or *haut mal.*

1. MINOR EPILEPSY.—The attack comes on *suddenly*, without warning, and is only of momentary duration, It is characterised by loss of consciousness, pallor (sometimes succeeded by trifling duskiness) of the face, a slight stare, and dilatation of the pupils. Any voluntary acts in which the patient may be engaged are abruptly stopped, while involuntary acts, such as walking, continue. He does not fall, and at most after a few seconds recovers himself, quite oblivious of anything having occurred. Occasionally after such an attack, certain curious automatic acts may be observed, or hysteroid convulsions may occur. The former are important from a medico-legal point of view, as in this condition criminal acts may be perpetrated, of which the patient has no knowledge, and over which he has no control. Thus articles may be stolen or hidden, or assaults committed, or the patient may indecently expose himself, and so on.

2. MAJOR EPILEPSY.—The attack is often preceded by premonitory phenomena, known as the *epileptic aura.* The aura varies widely in character in different cases, but is remarkably constant for the same person. It

usually takes one of the following forms:—A movement or sensation of numbness or tingling in the extremity of a limb; epigastric discomfort; a choking sensation in the throat, giddiness, or some disturbance of sight or vision.

The *actual attack* or *"fit"* may be conveniently described in three stages. The *first stage* is often ushered in by a piercing shriek, which is followed by absolute and instantaneous loss of consciousness, in which the patient falls more or less violently. *Tonic* muscular contractions of certain parts now supervene, by which the hands are clenched, the limbs twisted, the head and eyes rotated to one side, and the face hideously distorted; the breath is held; the face becomes pale and then livid; the pulse at the wrist is excessively feeble; and the pupils though sometimes contracted at first rapidly dilate as cyanosis supervenes. This stage usually lasts from thirty to forty seconds.

The *second stage* begins abruptly with the return of the breathing, which is violent and irregular. Loss of consciousness continues. Clonic spasms replace the tonic spasm, causing the features to work, the eyes to roll, the jaws to champ, the tongue to be frequently bitten, the limbs and trunk to be more or less violently convulsed, and the pupils to oscillate. The mouth foams; the face is livid; profuse perspirations break out; the pulse is full, and the heart beats violently; and both urine and fæces may be involuntarily discharged. This stage subsides gradually, and lasts from one to three or four minutes.

The *third stage* is marked by a gradual return of consciousness. The muscular spasms cease; while the patient opens his eyes, perhaps tries to get up, and looks bewildered. He is very exhausted and mentally confused; and shortly after, he usually falls into a deep sleep which may continue for some hours. After the attack a large quantity of pale limpid urine may be passed, and sometimes temporary paralysis of the limbs is observed.

The fit may not be repeated, but occasionally two or more occur in succession. More often it occurs periodically at intervals of a week, a month, or a year. It may come on by night as well as by day. During the intervals the patient may feel quite well, but the general health is defective as a rule. The memory may fail, the mind become dull, or acute mania or dementia may supervene. A single fit is rarely fatal, but the indirect risks of epilepsy are considerable. The most hopeful cases are those which occur in the young, and are due to some ascertainable and removable cause, and come under treatment early.

The main points on which a *diagnosis* should be based are:—the infrequency of its first occurrence late in life, its sudden onset, the insensibility of the conjunctiva, the dilatation of the pupil, the frequency with which the tongue is bitten, the frequent occurrence of injury from falling, the subsequent sleep, the short duration, and the absence of severe or continued headache persistent paralysis and emotional display.

TREATMENT.—The history should be carefully inquired into, and treatment should be directed to any discoverable cause. The most useful medicinal remedy is undoubtedly bromide of potassium, which should be given in full doses (especially just before the time when a fit is expected) and for a long period. Some have recommended the addition of a small dose of laudanum. It should, however, be remembered that this treatment is liable to cause general depression, and at best it is rarely curative. It is therefore advisable to combine tonic treatment with it, such as arsenic or strychnia (to be given after meals, well diluted) and cod-liver oil. Should any troublesome eruption be caused by the bromide, which does not pass off on stopping the drug, it may readily be cured, as a rule, by a short course ot arsenic. Iron is rarely of service. Mercury and iodide of potassium are indicated when there is a syphilitic taint. In some obstinate cases, counter-irritation to the nape of the neck by a seton or blister may be tried. Rest, good nutritious food, and avoidance of stimulants are urgently necessary.

During a fit, but little can be done beyond loosening the clothing, and gentle restraint to prevent injury.

HYSTERIA.

Hysteria is a disorder characterised by disturbances or perversions of the nervous functions which cannot be referred to any substantive disease, and in which weakness of the will plays a prominent part.

Hysteria usually first appears at some time between puberty and twenty-five years of age, and chiefly (though not exclusively) in females. Of the causes of hysteria we know very little, but strong emotional excitement, certain sexual conditions, and absence of definite occupation, stand out, one or all as having an important influence in its development. There is no necessary connection between hysteria and sexual disturbance, but when we bear in mind the large share which such functions assume in the physiological constitution of the female, it is not surprising that any profound impression on the one should be associated with disturbance of the other.

The *symptoms* of hysteria may be conveniently arranged in groups, which may occur separately, or in every possible combination.

1. The "*hysterical fit*" is a convulsive paroxysm, of *gradual* onset, beginning with sobbing, laughing, or other emotional displays, and often associated with a feeling of a ball in the throat (*globus*), till perhaps, with a scream, the patient falls to the ground violently convulsed and apparently insensible. This continues for a while—often for some hours—when recovery takes place with more sobbing, &c., and a feeling of exhaustion. The insensibility is very rarely complete; the convulsions are never tonic; the eyelids are closed and tremulous, and the pupils respond to light; the patient does not bite her tongue, and rarely injures herself; there is no foaming at the mouth; the fit is not followed by coma; and it never comes on during sleep.

2. *Mental disturbances.*—The controlling power of the will is always much weakened, and thus the emotions, relieved from proper restraint, are readily excited by trivial causes. In other cases there is absolute moral perversion, together with extraordinary whims and caprices; also a morbid self-consciousness, and a great anxiety to create interest and sympathy at any cost, which often leads to most outrageous acts of deception. Not infrequently hysterical patients take to drinking in excess.

3. *Sensory disturbances.*—These consist of (*a*) *neuralgic pains* with tenderness on pressure in various parts, which are often limited to a single part, *e.g.*, the scalp (*clavus hystericus*), the breast (*mastodynia*), the spine, the ovaries, or the joints; (*b*) various *perverted and morbid sensations*, as noises in the ears, flashes before the eyes, tingling in the limbs, or a peculiar sense of choking or constriction, as if from a ball in the throat (*globus hystericus*); (*c*) *exaggerated sensibility* (*hyperæsthesia*)—thus, one or more of the special senses may be excessively acute, or there may be intolerance of light and sound,

or the skin may be so sensitive that no part can be touched without causing pain; or (*d*) *anæsthesia* of the skin (generally limited in extent and irregularly distributed, but occasionally hemiplegic or paraplegic in distribution), or of the special senses. In most cases sensibility to pain alone is lost (*analgæsia*), while in other cases tactile sensibility is lost, impressions of temperature remaining unaffected; but all sorts of irregularity obtain in this respect.

Motor disturbance. Spasm.—In addition to the convulsions already described, we may meet with tonic spasm causing, for instance, more or less persistent flexion of the arm, spastic extension of the leg, or trismus. Tremor and rhythmical movements of the limbs are sometimes observed. *Paralysis.*—This is of very common occurrence, and may appear as hemiplegia or paraplegia, or be limited to a particular region, as in aphonia from paralysis of the laryngeal muscles. Hysterical paralysis may usually be distinguished by the history of the case; its liability to follow a hysterical fit; its incompleteness; its tendency to go and come suddenly and to shift its seat; the maintenance of normal electric irritability; its frequent association with complete anæsthesia, or with persistent rigidity from the beginning; and the absence of bed-sores or cystitis. The face is never paralysed in hysterical hemiplegia.

General functional disturbances.—The stomach is most often affected, giving rise to obstinate vomiting flatulence or painful dyspepsia; or the bowels may be persistently constipated or (more rarely) relaxed; or enormous quantities of pale limpid urine may be passed. Retention of urine is of common occurrence, but incontinence never happens. Palpitation, hurried irregular respiration, and menstrual derangements are also frequently observed.

Treatment.—Firm and considerate behaviour, assisted perhaps in an emergency by a douche of cold water, will generally suffice to rouse the patient from the fit. In bad cases chloroform may be given.

During the intervals between the fits the great aim should be to get the patient's confidence and respect, the moral effect of which will often alone determine a cure, and will always enhance the favourable influence of any other treatment which may be employed. In severe cases, it is of the utmost importance to remove the patient from her usual surroundings, and to transfer her from the care of her friends to that of a professional nurse. For all kinds of undue excitability, bromide of potassium is very useful, and may be combined or not with Indian hemp or valerian; for paralysis—strychnia, phosphorus, and faradisation; for anæsthesia—faradism with the wire brush. The importance of attention to the general health and mode of life cannot be overestimated. As a general rule, alcoholic stimulants do more harm than good to hysterical subjects.

HYPOCHONDRIASIS.

Hypochondriasis is an obscure mental condition which is most common in men of middle age. The hypochondriac pays such extensive attention to his bodily functions that he becomes a prey to his own feelings, and believes himself to be the subject of any or every conceivable disorder, real or imaginary. For the most part he has a healthy appearance, but sometimes he is languid, sallow, and the victim of dyspepsia, or constipation. The general plan of *treatment* resembles that for hysteria. The best thing is to get him to take up some definite occupation, which will serve to divert his attention from himself and his feelings.

CHOREA.

Chorea is almost exclusively a disease of childhood, rarely occurring before six or after twenty years of age. It, however, sometimes affects young females in pregnancy, and isolated cases are met with in advanced life. It is most common in females. A peculiar association exists between chorea, acute rheumatism, and heart disease. Thus (1) chorea often develops in the course of acute rheumatism, and *vice versâ;* (2) many choreic patients have previously suffered from acute rheumatism with or without complication; and (3) cardiac defects are common in chorea independently of rheumatism. Among the chief exciting causes may be mentioned *fright*, violent excitement, worms, menstrual troubles, and pregnancy.

Little is definitely known respecting the pathology of chorea. No constant anatomical change has been found in the central nervous system. It is evidently a disorder of *movements* rather than of *muscles*, and there are good grounds for regarding the motor area of the cerebral cortex as the primary seat of the disorder, but as to the nature of the latter, we have no exact knowledge. As to an adequate explanation of its causation we are almost equally in the dark. Nervous instability undoubtedly constitutes an important predisposing cause; and the frequent connection of chorea with acute rheumatism and endocarditis, taken together with other considerations, seems to point to some morbid state of the blood as a possible factor.

Symptoms.—Chorea usually comes on insidiously. The child is noticed to be restless and fidgetty, awkward in any movements requiring delicacy of adjustment, apt to jerk the limbs, make grimaces, or to drop things from the hand. There may also be some slight mental defect and emotional excitability. The movements are at first slight, and limited to some part, as the face or arm; but they afterwards become more violent and often involve the whole body. The condition is then very characteristic. In a well-marked case the head, trunk, and limbs may be twisted and jerked about in every conceivable form, but these movements differ from true convulsions (as has been already pointed out) in being moulded, as it were, to definite purposive acts. Thus the features work, the eyes roll, the lips are in turn pursed separated and drawn out; the tongue is now and again thrust from the mouth; the head is jerked from side to side; the limbs, especially the arms, are variously and irregularly flexed, extended or abducted, so that walking or even standing may become impossible; and the trunk may be tossed about so violently that the patient cannot be kept in bed. Food cannot be taken at all, or only with difficulty. The diaphragm shares in the general commotion, and the breathing is irregular and unequal. Speech is difficult or prevented by the movements of the lips and tongue. These movements are intensified by any attempted voluntary act, by excitement, or when watched; but they cease during sleep. Chorea may be limited to the face, a single limb, or to one side (hemi-chorea), or may involve the whole body. The affected muscles are always weakened, and actual paralysis of one or more limbs may supervene. The expression is always blank and fatuous, and in cases of long duration there may be actual mental defect.

With regard to the general health, the appetite is bad or capricious, the bowels are confined, and nutrition is impaired.

Anæmia rapidly develops in a severe case. There is no fever. Cardiac murmurs are very frequently observed, and present one or more of three varieties, viz., basic murmurs of hæmic origin, murmurs due to mitral regurgitation from atonic relaxation of the ventricular wall, and murmurs due to endocarditis.

The average duration is from four to six weeks. Chorea is rarely fatal except indirectly, as the result of exhaustion from inability to take food, or from bed-sores due to friction. Relapse and recurrence, however, are extremely common.

Treatment.—Rest in bed and strict avoidance of excitement are absolutely essential in all but the mildest cases. Any discoverable source of irritation should be removed if possible. A brisk purgative is useful at the outset.

The drugs employed may be divided into two classes, viz.:—*sedatives*, as chloral and bromide of potassium, and *nervine tonics*, as arsenic, zinc, iron, and silver. In acute severe cases it is well to bring the patient thoroughly under the influence of chloral for some few days, giving food in the short intervals between sleep. It should be remembered that acute chorea is a depressing and exhaustive disease, and a liberal supply of nutritious food and alcoholic stimulants is not only desirable but essential; and if the requisite amount cannot be given by the mouth, it must be supplemented by nutrient enemata. For a similar reason, chloral may be too depressing in the worst cases, with rapidly progressive cachexia, and under these circumstances, periodically repeated anæsthesia with chloroform may be necessary. As improvement takes place, the sedative must give place to a tonic plan of treatment; and in the milder cases, remedies of the latter kind are alone necessary. Of these, liq. arsenicalis in full and increasing doses is the most useful, either alone or combined with steel wine or the ammonio-citrate of iron. In chronic cases cod-liver oil, general tonics, good food, and change of air are of most service. When paralysis occurs, friction and faradisation of the limb should be employed and the patient encouraged to use it.

SUN-STROKE.

Sun-stroke, or insolatia, is the term applied to nervous disturbance arising from prolonged exposure to a high temperature. The temperature of the body is practically constant, and this uniformity is due to equilibrium between heat-formation and heat-loss. If, by the direct or indirect influence of the sun's rays, more heat is produced than can be got rid of by the ordinary channels, sudden elevation of the body temperature occurs, which is the immediate cause of the symptoms which characterise sun-stroke. This effect will be obviously enhanced by heavy or tight clothing, severe exertion, or deficiency of drinking-water.

Symptoms.—Not infrequently the onset is sudden, the patient falling down insensible and collapsed, with gasping respiration, extreme feebleness of pulse, and *elevation of the temperature.* This form is sometimes rapidly fatal.

More often the onset is less abrupt, and the attack sets in with exhaustion, nausea, restlessness, a high temperature, and the usual febrile symptoms. In severe cases death takes place from coma, perhaps ushered in by delirium or mania, or more rarely by convulsions, and it is rarely deferred between twenty-four to forty-eight hours. Even should recovery take place, troublesome nervous symptoms or confirmed epilepsy may subsequently appear.

Treatment.—The early employment of cold water is of the first importance, either in the form of a cold bath, a douche to the head, or a wet sheet pack. The bowels should be freely opened by enemata, and good nourishment is afterwards necessary.

NEURASTHENIA.

It is easy to criticise the use of this term from a strictly pathological point of view, but at the same time it is convenient to have some such title to apply to the heterogeneous symptoms arising from nervous exhaustion and debility. As the result of nervous over-strain, not only are all the functions of the body depressed, but the nerve-centres themselves become morbidly sensitive to disturbance from insignificant causes, and their physiological relation to each other becomes weakened and perverted. The condition thus brought about has often close resemblance to hysteria, but the disturbed nervous balance in the latter is rather the result of an ingrained faulty nervous organisation and has no necessary relation to debility; but in neurasthenia the primary nervous organisation may be perfectly normal, and the disturbed balance is the result of exhaustion and faulty nutrition. Neurasthenia sometimes affects the whole nervous system, and sometimes one part in particular. It is quite as common in men as in women.

An exhaustive description of the symptoms will not be attempted here, but among the most common are:—General weakness and irritability, disinclination for exertion, difficulty in mental concentration, weak memory, sleeplessness, and headache; extreme tenderness of certain parts of the spine, with pain radiating in various directions (*irritable spine*), numbness and tingling of extremities, emaciation, anorexia, gastric troubles, constipation; breathlessness, palpitation, weak circulation (cold hands and feet).

For this condition there is only one remedy—rest for body and mind, with abundant nourishment. Massage and electricity are most useful adjuvants in properly selected cases. The symptomatic treatment necessarily varies according to circumstances.

NEURALGIA.

The term neuralgia is applied to pain seated in the course of a nerve. It may arise from exposure to cold; from injury of, or pressure upon a nerve; or from direct implication of a nerve in some morbid process; but very frequently no adequate cause can be discovered. It is especially apt to occur in those of a nervous or hysterical temperament, and in those who are debilitated from any cause. Its pathology is unknown.

Symptoms.—Wherever seated, neuralgia presents certain characteristic features. The *pain* may be variously described as aching, boring, shooting, burning, &c.; but *it is always more or less paroxysmal.* It is generally severe, and sometimes of horrible intensity. On palpation, one or more *tender spots* are very constantly observed, with an invariable relation for each nerve, and they usually coincide with the exit of the nerve from a bony foramen, or its passage through some dense fascia. The pain is frequently associated (1) with local abolition of tactile sensibility; (2) with reflex muscular twitchings; (3) with congestion of the affected surface; and (4) with nutritive lesions of the skin, especially erythematous and herpetic eruptions. The pain of neuralgia is further characterised by its *tendency to shift* *from one spot to another*, in the course of the same or of some other nerve; and also by being *almost invariably unilateral*. As to the general health, neuralgia is *always attended with debility* from one cause or another, *e.g.*, mental anxiety, anæmia, gout, rheumatism, syphilis, and dyspepsia.

Neuralgia may attack any sensory nerve, whether it supplies the skin or the viscera. Special terms are employed to indicate neuralgia of special nerves: thus—tic douloureux (fifth cranial nerves); sciatica (sciatic nerve); brachialgia (nerves of the arm); gastralgia (nerves of the stomach); and so on. Separate description of each is here beyond our purpose.

TREATMENT.—Whenever possible the cause should be ascertained and an endeavour be made to obviate or remove it. Food should be abundant and nutritious, and should include a good proportion of fatty ingredients. It is scarcely necessary to add that rest, and freedom from worry, exhaustion or excitement are urgently indicated. To relieve the pain we may have recourse to internal or external remedies, or to both combined. Internally, the most important are—quinine (in large doses at first, followed by lesser ones); arsenic (♏ iij.-v. of the liquor three times a day); tr. of gelsemium (♏ xv.-xx. every four or six hours); sal ammoniac (gr. xv. three or four times a day); chloral or butyl-chloral; or alcoholic stimulants (best avoided if possible). Externally, hot fomentations, counter-irritation, systematic deep massage (*e.g.*, in cases of sciatica), with or without frictions with turpentine, camphor, belladonna or aconite liniments or veratria ointment, are useful. Should all these fail, hypodermic injections of morphia, or the local application of electricity (galvanism by preference) may be employed.

MIGRAINE.

Migraine is a peculiar paroxysmal form of headache, which is otherwise known as megrim, hemicrania, or sick headache. It is markedly hereditary, and usually first declares itself at some time between puberty and thirty years of age. A close atmosphere, intense heat, mental worry, excitement, fatigue, sexual excesses, and gastric derangement are common exciting causes. The immediate pathological cause of migraine is supposed to be a vaso-motor disturbance of some part of the brain, the effect of which is to cause irregular constriction and dilatation of the vessels; the remote cause consisting in a disturbance of the inhibitory and controlling functions of the brain.

Symptoms.—The attack begins somewhat suddenly with pain which is usually fixed to one spot, such as the temple, or one eye, from which it extends over the whole of one side of the head. The pain is commonly dull, sickening, and *throbbing;* but sometimes it is extremely sharp and violent. It is associated with tenderness of the scalp, a sense of heat about the head, and often with some injection of the eye and lachrymation. It is aggravated on lying down. Meanwhile the patient is pale, his extremities are chilly, he both looks and feels exceedingly ill, and the pupils are contracted. The pulse at the wrist is feeble, while on the affected side of the head it is strong. Anorexia is complete. After a while nausea and vomiting supervene, followed by sleep, after which he awakes much relieved. The duration of an attack rarely exceeds twenty-four hours. Relapses are of frequent occurrence. Migraine usually appears for the first time between puberty and thirty years of age, and disappears as a rule in old age.

The general plan of *treatment* is the same as that for neuralgia. Relief may be often afforded during a paroxysm by a cup of strong tea or coffee, by occasional doses of guarana (gr. xv.), bromide of potassium, or chloride of ammonium, or by the application to the head of cold compresses. Pressure on the carotid in the neck will sometimes temporarily stop the pain.

SUB-SECTION V.

TOXIC DISEASES OF THE NERVOUS SYSTEM.

ALCOHOLISM.

An excessive dose of alcohol behaves as an active poison and may immediately destroy life. But short of this, when taken in large quantity, or oft repeated, it is apt to induce more or less characteristic disorders of the nervous system and body generally, which will now be considered under the two heads of *delirium tremens* and *chronic alcoholism.*

DELIRIUM TREMENS.

True delirium tremens is the result of drinking alcohol in excess. Its onset is gradual. The patient becomes irritable and restless, he is wakeful and his sleep is troubled by horrid dreams, his volitional power is impaired, he loses his appetite, and his bowels are confined.

The actual attack is characterised by sleeplessness, delirium and tremor, with prostration and general functional disturbance. The mind is confused and excited, and soon begins to wander. The delirium is of a peculiar kind. It is of a busy garrulous type, the patient is suspicious of all around him, he sees all kinds of hideous forms, he fancies himself watched by enemies, or threatened by disaster. He is perpetually trying to get out of bed, and if not watched may leave the room or the house, or attempt to injure others, or to destroy himself. Occasionally he is wildly maniacal, while in other cases he is sullen and morose. The muscles of the limbs and body generally tremble and twitch, especially on the least attempt at voluntary movement. The body is prostrated, the face is pale or congested, the skin is clammy, the temperature is normal or only slightly elevated, the tongue is furred, the breath is fœtid; there is thirst, loss of appetite, and constipation; and the pulse is large, soft, and dicrotous. The above symptoms usually become worse towards night.

Most cases terminate favourably about the third or fourth day, when the patient passes into a sound sleep from which he awakes with a clear mind, but still very feeble. A marked rise of temperature, general and persistent trembling, and great prostration are very unfavourable signs.

It should be borne in mind that slight febrile disturbance, especially traumatic fever and pneumonia, are very apt to determine an attack of delirium tremens in habitual drinkers.

Treatment.—Alcohol should be absolutely prohibited in any form; liquid nourishing food should be given frequently; and the bowels should be regulated. Though by no means necessary in all cases, yet great benefit is sometimes derived from the employment of a medicinal sedative. Opium is preferable to chloral, and should be given in a fairly full dose in the first instance, and not repeated unless really necessary. The hypodermic injection of morphia is the best method.

Personal supervision by a competent attendant is far superior to any form of mechanical restraint—which should be avoided if possible. When there is great excitement, a tepid wet-sheet pack is sometimes of service; and while soothing the nervous system, it acts as a means of restraint. During convalescence, quinine and other tonics are indicated.

Chronic Alcoholism.

Among the chief results of chronic alcoholism we find obesity or emaciation, flabby and congested features, suffusion of the conjunctivæ, with a liability to various forms of acne. The appetite is bad, especially in the morning, the tongue is foul, the breath fœtid, with water-brash and other indications of digestive derangement. The implication of the nervous system is manifested by volitional weakness, emotional susceptibility, trembling, which is worse in the early morning and on movement, restless and troubled sleep, various forms of headache and vertigo, with disturbances of vision and hearing.

Later on, structural changes in various tissues and organs may supervene, especially in the liver, kidneys, and nervous system. It should also be recollected that the power of resisting morbid influences of any kind is greatly impaired in habitual drinkers, and thus intemperance becomes an important element in the prognosis of all diseases.

LEAD POISONING—PLUMBISM.

Though the susceptibility to the influence of lead varies much in different individuals, the symptoms to which it gives rise are tolerably characteristic when once they have developed. The condition is of frequent occurrence in painters, plumbers, glass-cutters, lapidaries, and others whose employment brings them into habitual contact with lead in some form or other. Drinking-water also, which has been stored in leaden cisterns, or conveyed through leaden pipes, may become the vehicle of contamination.

The subjects of lead-poisoning are pale, sallow, and as a rule ill-nourished, with a liability to digestive disturbances. The *gums exhibit a peculiar blue line* at their margins adjoining the teeth, due to the precipitation of sulphide of lead by sulphuretted hydrogen, which is generated by decomposing matters accumulating round the teeth. Attacks of *colic* characterised by severe griping pains in the abdomen, with obstinate constipation, are of frequent occurrence. The most common form of nervous disturbance consists of a paralysis of the extensor muscles of the forearm, giving rise to a *dropping of the wrist*. As a rule both fore-arms are affected; the muscles rapidly waste, forming a hollow on the back of each fore-arm; and their electro-contractility is quickly impaired and ultimately lost. The cutaneous sensibility is not affected, though in some cases there may be tenderness and pain over the muscles in the early stages of paralysis. The other muscles of the fore-arm and even those of the upper arm are apt to become enfeebled. Sometimes, too, the lower limbs may be similarly affected, as well as muscles in other parts.

The duration of these symptoms varies greatly, and their recurrence is frequent. The paralysis if neglected is very apt to become obstinate and even incurable. Chronic lead-poisoning is a common predisposing cause of gout, and is often associated with chronic albuminuria.

Treatment.—Those who work with lead should be careful to cleanse their hands and nails after work, and the habitual use of lemonade with sulphuric acid is probably of service by converting any lead in the alimentary canal into an insoluble sulphate. In the treatment of lead-colic, it is usually sufficient to relieve the pain with opiates and hot fomentations, the bowels generally acting without purgatives in the course of a few days. Should an aperient be thought necessary an enema is preferable to anything else. A little castor-oil with opium is also a valuable formula. For the paralysis, local galvanism is the only effectual remedy. When taken in hand quite early, faradism will suffice; but if no reaction occurs by this method, the slowly interrupted constant current is far more useful. With a view of eliminating lead from the system, iodide of potassium should be freely administered, together with an occasional aperient draught in the morning, of sulphate of magnesia.

MERCURIAL TREMORS.

A peculiar tremor sometimes occurs in connection with chronic exposure to the influence of mercury, which supervenes gradually, usually beginning in the arms, and accompanied with numbness or tingling and pain in the joints. Afterwards they become worse, especially under the influence of any excitement, and then extend to all the voluntary muscles of the body except those of the eyeball. All voluntary movements thus become uncertain, jerky, spasmodic, and feeble, until at last the patient is quite helpless. The tremors cease in the recumbent posture, and during sleep, except at a very advanced period of the disease. After a while, cachectic symptoms supervene, which are sometimes accompanied with grave nervous disturbance. There is no necessary association with mercurial stomatitis or salivation, though they are present, or have previously occurred, in a majority of cases.

If the patient be withdrawn from contamination sufficiently early, the progress is generally favourable in the course of a few weeks. Iodide of potassium, with quinine, iron, or other tonics may be given with advantage.

SECTION IX.

DISEASES OF THE SKIN.

TABULAR CLASSIFICATION.

Parasitic Affections	Animal	Scabies. Phiriasis.
	Vegetable	Ringworm. Favus. Pityriasis versicolor.
Glandular Affections	A. Sebaceous-glands 1. Faulty secretion	Seborrhœa. Acne punctata.
	2. Inflammation	Acne vulgaris. Acne sycosis. Acne rosacea.
	B. Sweat-glands 1. Quantity of secretion	Hyperidrosis. Anidrosis.
	2. Quality of secretion	Bromidrosis. Chromidrosis.
	3. Retained secretion	Dysidrosis. Sudamina.

Neurotic Affection		Pruritus.
Inflammatory Affections	Erythematous	Erythema.
		Urticaria.
	Papular	Lichen.
		Prurigo.
	Vesicular	Herpes.
		Cheiro-pompholyx.
		Pemphigus.
	Pustular	Impetigo.
	Squamous	Psoriasis.
		Pityriasis.
	Multiform	Eczema.
Hypertrophic Affections	of Pigment	Lentigo.
		Chloasma uterinum.
		Melanoderma.
		Morbus Addisonii.
		Nævus pigmentosus.
	of Epidermis and Papillæ	Tylosis.
		Clavus.
		Cornu cutaneum.
		Verruca.
		Verruca necrogenica.
		Ichthyosis (Xeroderma).
	of Connective-tissue	Scleroderma.
		Morphæa.
		Elephantiasis.
		Dermatolysis.
Atrophic Affections	of Pigment	Albinismus.
		Leucoderma.
	of Corium	Atrophia cutis.
	of Hair	Alopecia.
		Trichorexis nodosa.
New Formations	of Epidermis	Molluscum.
	of Connective-tissue	Keloid.
		Fibroma.
		Xanthoma.
	of Granulation-tissue	Lupus.
		Scrofuloderma.

PARASITIC AFFECTIONS.

ANIMAL PARASITES.

SCABIES.—This highly contagious disease is produced by the *acarus scabiei.* The eruption is essentially caused by the female *acarus*, which penetrates the epidermis, forming a furrow or tunnel in which it lays its eggs. The male lives on the surface and does not burrow.

From the irritation set up by the parasite, and by scratching, vesicles papules and pustules are formed, grouped together in every variety. It is attended with very severe itching which is worse at night, when warm in bed. Its usual distribution is in the flexor aspects of the limbs, front of the wrists, between the fingers and toes, palms of the hand, soles of the feet, genitals, and buttocks. In the vast majority of cases it will be found that a discrete eruption on the sole of the foot or the palm of the hand is due either to scabies or to syphilis.

The *treatment* consists of thorough scouring of the skin with flannel soap and hot water, followed by inunction with sulphur ointment. When the skin is very irritable, the application of mild Peruvian balsam or carbolic acid ointment is preferable. It should be remembered that sulphur ointment if applied too vigorously may induce an eczematous eruption, and it must therefore be used with discrimination. The general health often also requires attention, especially in the pustular forms of the eruption. All clothes and linen ought to be disinfected.

PHTHIRIASIS.—The human skin is attacked by three kinds of lice: (1) Pediculus capitis, limited to the head; (2) Pediculus corporis—to the trunk and limbs; and (3) Pediculus pubis—to the hair of the genitals, and occasionally the beard and axillæ.

Pediculosis capitis.—In this variety the pediculus is thin and slender, and is shorter than the P. corporis, and narrower than the P. pubis. Lice on the head cause intense itching, and secondary changes are produced by scratching. The scalp sometimes becomes covered with a semi-purulent discharge, which dries into thick hard scabs, from beneath which discharge oozes. The hair is matted. The glands of the neck also frequently become enlarged from the irritation.

Pediculosis corporis.—The body-louse is the largest of the three kinds, and its presence on the body causes great itching, and by scratching the skin is torn, and blood exudes, and forming crusts in the line of the scratch, a characteristic of the disease. Papules and urticarial wheals are also produced, and occasionally boils. As a result of long-continued scratching and inflammation the skin becomes pigmented and thickened. Usually the lice are not found on the surface, but are hidden away in the folds of the vest or shirt. The parts attacked are the shoulders, back, abdomen and thighs.

Pediculosis pubis.—The so-called crab-louse is shorter and broader than the other varieties. It is of a grey colour, and adheres to the hair close to the skin, causing great itching; and, like the head-louse, leads to scratching, which may give rise to secondary eczema. It is sometimes found in the axillæ, beard and eyelashes.

Treatment.—This consists in destroying the pediculi and their ova, and allaying the irritation. When pediculosis capitis occurs in children the hair should be cut quite short, the scabs removed by oiling, and the part freely washed, either with petroleum, carbolic acid lotion (1-20), boracic acid lotion, or other germicide wash, and the scalp should then be dressed with a mild mercurial ointment. In pediculosis corporis the clothes should be baked and the whole body washed with soap and water, and afterwards anointed with a mild antiseptic ointment such as white precipitate, sulphur, stavesacre, or tar. Treatment of an analogous kind is required for pediculosis pubis.

VEGETABLE PARASITES.

There are three varieties of vegetable fungi which produce three distinct diseases of the skin. They are (*a*) Tricophyton; (*b*) Achorion Schönleinii; (*c*) Microsporon furfur.

(*a*) Tricophyton produces ringworm of the body (*tinea circinata*); ringworm of the scalp (*tinea tonsurans*); ringworm of the beard (*tinea sycosis*); *eczema marginatum;* and ringworm of the nails. (*b*) Achorion Schönleinii produces *favus*. (*c*) Microsporon furfur produces *pityriasis versicolor*.

TINEA CIRCINATA commences as a small erythematous patch, and spreads in the form of a ring, or coalescing with other rings becomes gyrate in outline. The margin is papular or vesicular, while the centre becomes pale discoloured and branny. Epidermis scraped from the margin, and treated with weak solution of caustic potash shows the characteristic fungus, which consists of spores, arranged more or less in lines, and fine mycelium threads forming a network between the epidermic scales. This disease occurring in the axillæ and in the inner sides of the thighs is accompanied by severer inflammation, especially in hot climates, and forms the so-called *eczema marginatum*, which is often difficult to cure.

TINEA TONSURANS attacks the scalp, and is most common in children. Its different appearance on the scalp is due to the anatomical structure of the part. Before the fungus has penetrated into the hair follicles the disease may easily escape notice. The hairs when affected break off short, and become dull-looking, twisted and extremely brittle ; and, if extracted and placed in weak liquor potassæ, show large quantities of spores lying in rows, and also some mycelium. Not unfrequently it is complicated by suppuration. When the latter is superficial it may be mistaken for *impetigo*, and when deep-seated it forms the condition known as *kerion*. Usually, however, it becomes dry and chronic. A patch may become so bald as to be mistaken for alopecia areata. Diffuse ringworm is most difficult to diagnose and to cure. Ringworm attacking the beard, moustache, or eyebrows is known as *tinea sycosis*.

Ringworm may attack the nails causing them to become distorted, brittle, broken and lustreless.

FAVUS OR TINEA FAVOSA commences as a small erythematous patch which gradually spreads in the form of a ring, and becomes covered with cup-shaped sulphur-coloured crusts—a characteristic of the disease —which emit a mouse-like odour. The crusts are attached firmly to the skin, and when removed leave pits and sometimes scars of varying size, as a result of long-continued pressure. The crusts are dry and brittle and composed of the mycelium and spores of *Achorion Schönleinii*. Its favourite seat is the scalp, but no part of the body is exempt.

PITYRIASIS VERSICOLOR is characterised by fawn-coloured, slightly scaly, irregular patches which are especially common on the front of the chest. Spores and mycelium may be readily detected on microscopic examination of the scales, when scraped off and treated with liquor potassæ.

Treatment.—T. circinata and pityriasis versicolor may be easily cured by washing with soft soap and subsequent dressing with sulphurous acid, carbolic lotion, or weak mercurial ointment. T. tonsurans when quite recent is generally amenable to painting with tincture of iodine, or blistering with acetum cantharidis. In chronic and diffuse ringworm, the head should be shaved and well washed with soft soap two or three times a week. Carbolised oil (1 in 20) should be used daily to the whole scalp. As to special parasiticides, perhaps the most useful is oleate of mercury (5 per cent. to 10 per cent. strength); but the same remedy will not suit all cases, and a selection of formulæ is given in the therapeutic index. In obstinate cases, epilation should be practised. In kerion the inflammation suffices to destroy the fungus, so only soothing applications are needed. Favus should be treated in the same way as ringworm.

GLANDULAR AFFECTIONS.

SEBORRHŒA.—This depends upon an increased secretion of sebaceous matter, and exhibits two varieties—S. oleosa, and S. sicca.

In *S. oleosa* the fatty portion of the sebum is in excess giving the skin a greasy appearance. It forms dirty-looking greasy patches, which are easily detached, and without any inflammatory redness, unless complicated by eczema. It is most common on the face. In *S. sicca* the secretion is far less oily, and sometimes consists only of dry scales. It most frequently affects the scalp, forming "scurf," but may occur on other parts. The skin becomes dry, harsh, and tight, and the hair falls out.

Treatment.—Careful regulation of all internal organs, combined with suitable tonics. After cleansing the surface, precipitated sulphur ointment (ʒ ss. to vaseline ℥ j.), mild white or red precipitate ointment, weak carbolic or tar lotions, or glycerine and water with a little spirit may be employed locally. Strong applications should be avoided. In the oily form, spirit lotions—with or without tar—are of great service.

Acne punctata.—This term includes *Comedo* and *Milium*. *Comedo* is simply an accumulation of sebaceous matter in a patent gland on the face, chest, and back, appearing as a small black or yellowish spot. A minute living grub called *Acarus folliculorum* is occasionally contained in the substance which can be squeezed from the follicle. The dark speck is due chiefly to dirt adhering to the oily matter. Comedones form at or about puberty commonly, but they are sometimes congenital.

Treatment.—The part should be washed with hot water, freely rubbed with equal parts of soft soap and spirit, the comedones squeezed out and sulphur or mercurial ointment be applied. In obstinate cases scarification may be employed.

Milium is a distension of a sebaceous gland with sebum without any external opening. The local treatment should consist of an incision, removal of the contents, and slight scarification or scraping of the cavity.

Acne vulgaris.—This consists of inflamed sebaceous glands and surrounding tissue. A comedo or milium may be simply hyperæmic, or the inflammatory process may run on to a papule, tubercle, or pustule, or even give rise to a boil. Different varieties are described, according to the stage. A. papulosa, A. tuberculosa, A. indurata, and A. pustulosa. In severe old-standing cases, scars are often left. It may be caused by the action of drugs, both internal and local; hence the names *bromide*, *iodide*, and *tar acne*.

Treatment.— Strict attention to personal hygiene, fresh air, exercise, and plain food are of the first importance. Tonic aperients, especially magnes. sulph. with iron, and quinine, are often beneficial. Arsenic, iron, mineral acids with vegetable bitters, and sulphide of calcium are each of service in certain cases. The local treatment varies according to the amount and nature of the inflammation. In quite mild cases it is essentially the same as that advised for comedones.

ACNE SYCOSIS attacks only hairy parts, more especially the beard and moustache, and forms papules or pustules, through each of which a hair passes. When pustules coalesce, it forms a doughy mass somewhat like the inside of a fig—hence the term *sycosis*. It should not be confounded with ringworm of the beard. There is more or less pain and heat in the part; and the hair follicles may be destroyed, causing baldness. It is believed to be due to a specific micro-organism.

The *treatment* consists of antiseptic applications—mercurial or otherwise—varying in strength according to the amount of inflammation. In the later stages epilation is often of great service.

ACNE ROSACEA rarely occurs in early life, and is limited to the face. It begins with flushing of the cheeks or nose, ultimately followed by permanent vascular dilatation, and often accompanied by acne vulgaris and seborrhœa. In severe cases the veins may become large and tortuous, and tuberous masses may form. Faulty digestion, feeble circulation, and alcoholism are frequent causes.

Treatment.—The general health requires close attention, and remedies directed to the improvement of digestion and circulation are especially indicated.

Locally, applications of sulphur, perchloride of mercury, and carbolic acid are most useful, graded in strength according to the amount of inflammation. In aggravated cases much relief may be given by free scarification of the dilated vessels, or by electrolysis.

DISEASES OF SWEAT GLANDS.

Hyperidrosis—increased sweating. *Anidrosis*—deficient sweating. *Bromidrosis*—sweating with offensive odour. *Chromidrosis*—coloured sweat. *Dysidrosis*—An inflammation set up by disordered sweating, and possibly identical with cheiro-pompholyx. *Sudamina*—minute non-inflammatory vesicles looking much like drops of water on the surface of the skin, caused by profuse sweating, and possibly some qualitative alteration in the sweat. Minute inflammatory vesicles and pustules *(miliaria)* may arise under similar circumstances, and are often associated with sudamina. "Red-gum" or *strophulus* of infants is now regarded as essentially an inflammatory sweat-rash.

The *treatment* consists in strict cleanliness, frequent change of linen, and the local application of mild antiseptic washes or powders.

NEUROTIC AFFECTION.

PRURITUS denotes itching of the skin, and must not be confounded with prurigo. It may be general or local. General pruritus is common in aged people *(P. senilis)*, diabetes, jaundice, pregnancy, gout, &c. The most common local varieties are *P. ani*, and *P. genitalium*. *P. ani* usually arises in connection with piles or worms or profuse perspiration with decomposition of the sweat, or local organic disease. *P. genitalium* is most common in children as the result of ascarides, and in women, from uterine disorder, menstrual disturbance and diabetes; but often no cause can be discovered.

The *treatment* should be directed primarily to the removal of the cause wherever this is possible. The bowels require careful regulation, and the diet should be plain and unstimulating. Much relief to itching may often be given by external remedies. In general pruritus, alkaline baths, weak spirit lotions to which liq. carbonis detergens (1 in 40), or carbolic acid (1 in 60) may be added, menthol ointment (ʒ ss. to ℥ j. of lard), or camphor chloral may be tried. In P. ani weak ammoniated mercury or nitrate of mercury are useful. In P. genitalium saturated solution of boracic acid, tinct. benzoin. co., weak solution of mercuric perchloride (1 in 2000) or cocaine ointment may give relief. Sedatives may be necessary to procure sleep at night.

INFLAMMATORY AFFECTIONS.

Erythematous Inflammations.

Erythema consists of superficial red patches which disappear on pressure exactly like the early stages of erysipelas. It usually fades away in a few days, with slight desquamation. As a rule there is little or no constitutional disturbance or fever. The following varieties are met with. *E. simplex*—arising from local irritation of any kind; *E. pernio*—or common chilblain; *E. intertrigo*—due to friction of opposed surfaces of tender skin; *E. læve*—the redness so often occurring on œdematous parts, and sometimes followed by sloughing. *E. fugax*—red patches which come out suddenly on the face or trunk, only lasting a short time, and generally associated with gastro-intestinal irritation.

Erythema multiforme consists of reddish inflammatory patches, more or less elevated from œdema, generally symmetrical in the main, and attended with mild constitutional disturbance, and sometimes with rheumatic pains. The rash subsides in from 1 to 4 weeks, leaving a slight hæmorrhagic stain for a time. The back of the wrists and dorsum of the feet are most commonly affected. The patches are usually small and round *(E. papulatum)*, sometimes in small rings *(E. iris)*, sometimes in larger intersecting rings *(E. gyratum)*.

Erythema nodosum is a localised inflammatory œdema attended with extravasation of blood, and is allied to purpura. It consists of oval swellings which appear in crops, generally on the front of the legs, but sometimes on other parts of the body. The nodes are at first hard and tender, gradually becoming softer, but never suppurating. Their colour is at first bright red, afterwards more dusky, and as they disappear they pass through the phases of a bruise. The disease is usually attended with pains in the legs, fever, headache, and general malaise. It is most common in young girls. Recovery occurs in about three weeks.

Plain food, with tonics such as quinine and iron, in combination with an aperient is all that is required by way of treatment.

Urticaria is essentially characterised by the formation of wheals, which vary greatly in size and shape, and are attended with itching or stinging sensations, hence the common term *nettle-rash*. There is usually but little constitutional disturbance. It may be either acute or chronic. It usually arises from direct irritation of the skin by exposure to cold, by scratching, or by the bites and stings of insects; from reflex irritation set up by worms or uterine disease; from certain kinds of food, especially shell-fish; from the administration of certain drugs, such as copaiba; or from gastro-intestinal disorder.

Treatment.—In acute forms, an emetic or a saline aperient is indicated. The diet should be easily digestible and non-stimulating. In chronic forms, the general condition requires close investigation, especially the digestive functions, the correction of which should be aimed at. Tonic remedies and change of air are often needed to complete the cure. Locally alkaline or spirit-lotions may be used to relieve the itching.

PAPULAR INFLAMMATIONS.

LICHEN exhibits two varities:—*L. planus* or *ruber*, and *L. marginatus*. *L. planus* consists of small irregular-shaped flattened papules, covered with thin scales, and varying in size from a pin's head to a split pea. The papules are of dusky red colour, and though at first discrete, are apt to merge together, forming larger patches. On its disappearance, the skin remains stained for a long time. It is attended with more or less itching. It runs a tedious course, and relapses are frequent. Its usual seat is on the flexor surfaces of the limbs, but no part is exempt, it is symmetrical, and is most common in middle and advanced life.

L. marginatus consists of small red papules, grouped in rings or segments of rings, and generally limited to the trunk of the body. It grows by extension of the rings and runs a very tedious course. As the papules fade the skin is left stained. It is commonest in childhood.

Treatment.—For *L. planus* the best internal remedies are arsenic combined with nervine tonics, and generous feeding. Repeated small doses of hydrarg. perchlor. sometimes relieve the itching, and in some chronic cases the waters of Bath or Harrogate are useful. Locally, if the skin be irritable—alkaline baths, lead lotion or ointment; but in chronic cases—mild tarry applications or chrysarobin ointment gradually increased in strength. For *L. marginatus*, good nutritious feeding, fresh air, cod-liver oil, and the local application of any simple emollient are usually sufficient to cure.

Prurigo.—This affection is characterised by pale slightly elevated papules, generally seated on the trunk, and attended with great irritation which leads to scratching. The apices of the papules are thus torn off, leaving little hæmorrhagic points behind, the appearance of which is characteristic. It is often associated with urticaria, and as a primary affection is most common in delicate children, but it also occurs as a result of the irritation set up by parasites, &c.

For the prurigo of children, good hygiene, liberal feeding and cod-liver oil are generally of most service, with the local application of alkaline lotion, or tarry preparations. The treatment of secondary prurigo is that of the condition which gives rise to it.

Vesicular Inflammations.

Herpes is an acute inflammatory affection consisting of vesicles situated on an inflamed base. They are generally few in number, of the average size of a large pin's head, with clear serous contents, which soon become turbid, and dry up in a few days into a scab. Its chief varieties are: *H. febrilis*—characterised by one or small groups of vesicles on the lips, nose, ear, or buccal mucous membrane, causing some smarting pain. It is a common result of catarrh, pneumonia, cerebro-spinal and other fevers. *H. progenitalis*—occurring on the prepuce, glans penis, or in the female on the labia of the vulva, and mainly dependent upon local irritation. *H. zoster*, *zona*, or *shingles* is peculiar in following the course of some cutaneous nerve, generally one of the intercostal nerves, but it may attack the face, belly, shoulder, or thigh. It is almost always unilateral in position, and is accompanied by neuralgic pains which are apt (especially in the old) to remain long after the eruption has disappeared. It never relapses and seldom recurs.

The *treatment* of herpes is, as a rule, quite simple. Locally some soothing or anodyne ointment is often all that is necessary. In zoster the part should be fomented with hot water, followed by the application of starch powder with a little oxide of zinc and camphor, and the surface should be protected from the air and external injury by cotton-wool secured by a bandage. Sometimes the pain is so severe as to require a subcutaneous injection of morphia. The general health needs close attention, and tonic remedies — especially quinine, arsenic and iron—are of great service.

Cheiro-pompholyx.—A disease consisting in the formation of vesicles or bullæ with little or no surrounding inflammation, limited almost entirely to the hands and feet, often to the former alone, and usually symmetrical, running an acute course, and relapsing with a variable interval. The nails are often affected. Believed to be of neurotic origin, from depression and worry.

Treatment.—Change of air, and tonics; locally, soothing remedies to relieve the itching.

Pemphigus.—There are two varieties: *P. vulgaris* and *P. foliaceus*, the latter being rare. *P. vulgaris* commences in the formation of oval or round bullæ of various sizes, the fluid in which is at first clear and alkaline, and afterwards turbid and acid, situated on slightly inflamed bases scattered irregularly on the body, usually followed by successive crops. All parts of the skin and mucous membrane, except the scalp, palms and soles, may be attacked. Bullæ may dry up, leaving some pigmentation, or burst, leaving ulcers. There may be much irritation, and, though constitutional symptoms are usually slight, want of sleep may wear the patient out. *P. foliaceus* is a very severe variety in which bullæ form and burst with great rapidity and cover a large area. Raw patches are produced which have no tendency to heal.

Treatment should consist of arsenic in full and repeated doses, together with careful regulation of the bowels and good nourishment. A warm bath lasting for several hours, and daily dressing of ulcers with weak iodoform ointment or other antiseptic are very beneficial.

Pustular Inflammation.

Contagious Impetigo is a local disease produced by the inoculation of pus. The pustules so formed dry into yellow scabs which adhere firmly to the skin, but are not surrounded by inflammatory areolæ as in the case of pustular eczema. There is no ulceration as with a pustular syphilide. It may, however, be rubbed into an inflammatory sore, and in this condition constitutes what was formerly described as *ecthyma*. The constitutional symptoms are slight.

Treatment.—Removal of the scabs by means of antiseptic washes and dressing the surfaces with mild mercurial ointments, such as ung. hydrarg. ammon. (gr. x. to ℥j. of lard).

SQUAMOUS INFLAMMATIONS.

PSORIASIS is a common disease occurring in the form of white silvery scales on slightly infiltrated patches of skin. The patches tend to spread at their periphery and clear up in the centre, thus forming rings (*P. circinata*), which sometimes intermingle (*P. gyrata*).

The typical seats of psoriasis are the fronts of the knees, the tips of the elbows, extensor surfaces of the limbs, and the scalp; though it not infrequently affects the trunk. The patches vary in size, are dry, with well-defined edges. There may be itching, but it is often absent. The course is very chronic, and recurrence is very common. Hyperæmic and irritable patches are apt to become eczematous from scratching. Its cause is unknown, but nervous depression and exhaustion appear to be common predisposing causes, and some believe that gout is an important factor in many cases.

Treatment.—Arsenic internally is very useful. The local treatment consists in warm bathing daily, with soft soap, to remove scales, and the free inunction of ointments containing tar in one of its many forms. Chrysophanic acid is also very beneficial (2-10 grains to ℥j. of lard), care should be taken to keep it from the eyes and not to use it too strong for too long a time, as it may cause much inflammation in some patients. It discolours linen and therefore is best applied in traumaticine, which is a solution of gutta-percha in chloroform.

PITYRIASIS RUBRA.—A chronic scaly disease of skin, characterised by a general redness, which gradually spreads over the whole surface, giving rise to desquamation of large masses of epidermis. There is no infiltration or exudation. There is little constitutional disturbance at first, but later on the patient emaciates, and a fatal termination is the rule after a very chronic course.

Treatment.—Prolonged baths, application of oil, nourishing diet and tonics.

MULTIFORM INFLAMMATION.

ECZEMA.—The distinctive character of this affection is its versatility; commencing as an erythema, it may become in turn papular, vesicular, pustular, or scaly, with more or less pigmentation and infiltration. It is the commonest skin disease, and may occur on any part of the body, but chiefly affects those parts where the skin is thin. It may be acute or chronic; and it is very liable to recur. Local irritation of any kind, a varicose condition of the veins, dyspepsia, and gout may be mentioned as the most frequent causes of eczema, but the cause is often very obscure. A vast and unnecessary number of names have been applied to varieties of eczema, due to its temporary condition, its seat, or its assumed cause, but only a few of the most typical need be mentioned here.

Acute eczema begins with redness and swelling, followed by an eruption of minute vesicles which burst leaving a raw discharging irritable surface. From desiccation thin brownish scabs may form. The inflammation may then subside, or fresh crops may appear, passing through similar stages. Successive attacks may thus prolong the disease or it may pass into the chronic form. There is but little constitutional disturbance. *Infantile eczema* chiefly affects the head and face in young children. It is characterised by its tendency to be pustular, and to form thick yellow crusts.

The cervical and occipital glands are apt to be enlarged, and subcutaneous abscesses are frequently met with. It is specially common in strumous children. *Seborrhœic eczema* chiefly affects the back and chest in the form of well-defined thickened patches covered with greasy scales. *E. intertrigo* represents a further development of erythema intertrigo. *Chronic eczema* is characterised by indurated and irritable patches often covered with dry scales, or cracked. It is especially common about the lower part of the legs in old people of a gouty or rheumatic habit, or it may affect the hands and the feet.

Treatment.—This must vary according to severity of attack, and according to extent, position, and cause—when the latter can be made out. In a very acute and wide-spread attack, rest in bed may be necessary, light diet free from stimulants, regulation of bowels, and small repeated doses of vinum antimoniale. Locally, avoidance of all irritants, such as washing with water and soap, exposure to draughts, and irritating clothes. Remedies must be carefully selected. If the surface be oozing, dusting powder such as starch and oxide of zinc, or the former alone may be used; if burning, smarting, and drier, then calamine lotion is very useful, as also zinc and gelatine dressings. In the sub-acute and chronic forms, the diet should be generous, and tonics may be required. Any co-existing struma, gout or rheumatism, should of course be specially treated. Locally, stimulating and mild antiseptic remedies are required, but much skill is required in determining the strength that can be borne in each individual case. Lead, mercury, or tar ointments may be rubbed in to improve the circulation and promote absorption of the inflammatory products. In chronic indurated forms much can be done by massage, bathing, and firm bandaging. In the seborrhœic form mild sulphur, resorcin, and ichthyol preparations are of most service.

HYPERTROPHIC AFFECTIONS.

A. OF PIGMENT.

LENTIGO, EPHELIS, or FRECKLES.—The increase of pigment granules in small or large patches, chiefly on the face, neck, and hands, varying much in colour, becoming darker on exposure to light.

CHLOASMA UTERINUM.—A brown pigmented patch with a crescentic outline, usually seen on the foreheads of women with uterine disease, or as a result of frequent pregnancies.

MELANODERMA.—A term given to any pigmented patch, such as occurs round the nipples in pregnant women; or below the eyes, from ill-health.

MORBUS ADDISONII.—Refer to page 127.

NÆVUS PIGMENTOSUS.—Pigmentary mole is usually congenital: often covered with coarse hairs.

Treatment.—The success of local remedies varies much in different individuals. Mercurial lotions, such as hydrarg. perchlor. (gr. j. to ℥j. of spirit), and ointments containing mercury or sulphur, or salicylic acid are often most useful.

B. OF EPIDERMIS AND PAPILLÆ.

TYLOSIS or TYLOMA.—A thickening of the epidermis, usually occurring on the hands and feet. It arises from intermittent pressure or friction; due to occupation on hands, and on the feet chiefly to badly fitting boots.

Clavus.—A corn is a localised hypertrophy of the epidermis. There are two kinds, hard and soft. The hard corn is conical in shape, and the apex pressing down upon the corium causes atrophy, and so forms a pit. The exquisite pain experienced is due to pressure on the nerves in the papillary layer. A corn of either kind may give rise to a subcutaneous abscess.

Treatment.—Attention to the fitting of boots. Removal of horny substance by the knife, maceration with scraping or filing, or by glacial acetic acid. Soft corns should be dried by daily dusting with boracic acid, and by separating the toes by means of cotton-wool, and afterwards treating on the same principles as hard corns.

Verruca.—Warts differ in appearance according to situation, on exposed parts they are hard, on mucous membranes—soft. A hard wart may be simple or compound, involving few or many papillæ. They chiefly affect the face and hands. Soft warts are more pointed and vascular than hard warts. They occur in both sexes on the perineum, margin of anus, and on genitals. They are not necessarily of venereal origin.

Verruca necrogenica is the name given to a troublesome kind of wart which occurs on the hands of those engaged in post-mortem work.

Treatment—Either hard or soft variety may be snipped off with curved scissors and the base cauterized, or they may be destroyed gradually by caustics.

The numerous warts frequently accompanying seborrhœa will cease to appear and those already formed may entirely vanish on the cure of the seborrhœa with mild sulphur preparations.

Ichthyosis and Xeroderma are terms applicable to one and the same disease, the latter denoting a milder type than the former. True ichthyosis is congenital, and represents hypertrophy of the papillæ and epidermis. The condition of the skin varies from that of mere dryness with slight thickness, to that of hard dark-coloured plates resembling the skin of a crocodile. *I. hystrix* is a severe variety, in which the growth assumes the form of conical warty masses projecting half an inch or more above the surface. The secreting functions of the skin are greatly disturbed.

Much relief may be obtained by prolonged soaking of the part in water or weak alkaline lotion, followed by friction with soap, and inunction with oil or glycerine.

C. OF CONNECTIVE TISSUE.

SCLERODERMA.—A rather rare disease chiefly affecting the head, neck, and chest, and caused by hypertrophy of the connective tissue, which makes the skin hard and inelastic. When the disease is fully developed the skin has a waxy smooth appearance, something like ivory, and is often associated with more or less pigmentation. Occasionally it endangers life by interfering mechanically with the respiratory movement of the chest; but speaking generally, it tends to slow recovery. In some cases the skin afterwards becomes atrophied and shrivelled. It is most common among women.

Treatment.—Protection from cold, generous diet, tonics, cod-liver oil, with local massage, and regular application of galvanism.

MORPHŒA is a local form of scleroderma, occurring in well-defined patches or bands, affecting any part of the body, but especially the lower limbs.

ELEPHANTIASIS and DERMATOLYSIS are so rare in this country as not to call for detailed description here.

ATROPHIC AFFECTIONS.

A. Of Pigment.

Albinismus is a congenital absence of pigment from the skin, hair, iris, and choroid, which may be either general or local. Persons suffering from this condition are termed *albinos*.

Leucoderma consists of well defined white patches, of rounded shape, bordered by pigmentation in excess, which gradually merges in the healthy skin. They gradually enlarge, till only a few brown patches with concave edges are left to represent the normal skin. No treatment is necessary.

B. Of Corium.

Atrophia cutis. Atrophy may occur in elderly people *(atrophia senilis)*. It results also from great distension, prolonged pressure, and as a sequela of some skin diseases.

C. Of Hair.

Alopecia may be either congenital or acquired. It is usually a senile change, but it may occur prematurely from causes of a general character, such as fevers and severe illness; or from local causes, such as eczema, psoriasis, and especially seborrhœa. Ringworm and favus may cause baldness by destroying the hair follicles.

Alopecia areata begins with sudden falling out of the hair on a limited spot, the skin of which is quite smooth, polished, and distinctly thinner than normal.

On the edge of the patch, short hairs are found, bigger at their free ends than at the neck of the follicle, and for some distance around, the hairs are loose, and may be easily pulled out without pain. No micro-organisms have been proved to be the cause of the disease, and it is probably of a neurotic origin. It must be carefully distinguished from ringworm. The younger the patient, the more favourable the prognosis.

Treatment.—Internally—nervine tonics. Locally—stimulating applications, such as ammonia, cantharides, and (in obstinate cases) blistering,

TRICHOREXIS NODOSA is a term applied to a disease chiefly affecting the hair of the beard and moustache, and consisting of small swellings involving the whole thickness of the hair at intervals, with a tendency to break at these points.

NEW FORMATIONS.

A. OF EPIDERMIS.

MOLLUSCUM CONTAGIOSUM.—This disease consists of little sessile or pedunculated tumours, round in form, with a flat top, umbilicated, hard to the touch, white and shining, and (when fully grown) about the size of a pea. The tumours are composed of epithelial cells of the prickle layer. The disease is most common on the face, neck, breasts, and genitals, and is probably contagious.

Treatment.—Remove with curved scissors, or open with a knife and squeeze out the contents, or scoop out with a surgical spoon.

B. OF CONNECTIVE TISSUE.

KELOID.—This title no longer includes the limited form of scleroderma called morphœa, but refers to the keloid of Alibert, which is generally believed to arise only from scars. It first appears as an irregular shaped swelling, resembling an urticarial wheal, which grows at a variable rate into a fusiform or elongated mass, which (should it greatly extend) becomes stellate, with its processes tightly drawn down, looking claw-like, hence its name. The surface is usually pale and smooth. No part of the body is exempt from it. It may spontaneously disappear or last a lifetime. It is generally tender, and sometimes painful.

Scarification appears to be the best treatment.

FIBROMA occurs as pendulous tumours of various sizes, from that of a pea to that of a fist and larger. Single or multiple, they are usually attached to the skin by a narrow pedicle, but are occasionally imbedded in the substance of the corium. They are soft, elastic to the touch, of normal colour of skin; sometimes on their surface is seen the orifice of a large sebaceous gland. They may occur on any part of the skin, and occasionally so as to cause much inconvenience, in which case they may be removed by a surgical operation.

XANTHOMA.—There are two varieties:—*X. planum*, *X. tuberosum*. The former appears in flat, slightly raised, irregular-shaped patches of a chamois-leather colour, with sharply defined margins, and usually symmetrically placed on the upper or lower eyelids. *X. tuberosum* occurs in the form of small tubercles of a similar colour, scattered on various parts of the surface. If the new growth cause inconvenience it can be removed by the knife or scissors.

C. OF GRANULATION TISSUE.

LUPUS VULGARIS is a new growth occurring on the skin or mucous membranes due to micro-organisms closely allied, if not identical, with tubercle bacilli. It almost always first appears before puberty. It is most common upon the face. On the skin, it begins with apple-jelly coloured spots, gradually assuming the form of small tubercles, and by extension at last forms a dull red irregular patch or patches, covered with thin scales or scabs. It may remain quiescent for years, the central parts becoming atrophied and absorbed, leaving scars; or it may break down and cause destructive ulceration, which heals very slowly and leaves ugly scars.

The *treatment* consists in the removal of the tissue by scraping, actual cautery, or caustic applications; together with everything that may tend to improve the general health.

LUPUS ERYTHEMATOSUS consists of superficial reddish patches of various shapes. On the face—its favourite seat—it commonly assumes a butterfly shape, owing to its attacking the nose and cheeks symmetrically. The patches are well-defined, slightly raised, and covered with adherent scales, which on removal are found to dip into the sebaceous follicles. As the patches spread, their centres atrophy and gradually assume a cicatricial appearance. There are no tubercles, nor is there any ulceration. It is rarely met with before 20 years of age. Exceptionally, the disease runs an acute and dangerous course.

Treatment.—Soothing applications in the early stages. Later on when less irritable, thorough applications of equal parts of soft soap and spirit are useful. In obstinate cases linear scarification is recommended.

SCROFULODERMA.—This disease seems to be closely allied to lupus vulgaris, but differs in some clinical characters. Lupus is not necessarily connected with enlargement of the glands; whereas scrofuloderma usually begins in enlarged glands, or in scars resulting from a scrofulous abscess. The latter spreads more rapidly and ulcerates earlier, and the ulcers have undermined, flabby, bluish edges, with uneven bases.

Treatment.—Change of air, nourishing diet, tonics and cod-liver oil. Locally, scraping away all traces of the disease, or use of the cautery.

THERAPEUTIC INDEX.

Note.—In submitting the following therapeutic index, my intention has been merely to provide examples of prescriptions which have been found useful by myself and others, and to suggest in a general way the lines of treatment hitherto adopted. Any slavish adhesion to fixed formulæ is strongly to be deprecated as tending to stifle the spirit of original enquiry on the part of the practitioner, and taking the place of that which personal practice and experience alone can properly give. The suggestions made under each heading should be read in conjunction with the paragraphs in the text which relate to the same subject.

Acne.

1. Hot fomentations and application of equal parts of alcohol and soft soap.
2. Contents of follicles to be systematically squeezed out.
3. ℞ Ung. sulphuris (precip.) ʒi.-iv. to vaseline ℥i.
4. ℞ Sulph. hypochlorid. ʒi.
   Potass. carb. gr. x.
   Vaseline ℥i. ft. ung.
5. ℞ Ung. sulph. iodid. (half B.P. strength). Useful in severe forms.
6. ℞ Sulph. precip. ʒi.-iv.
   Glycerini ℥i.
   Aq. rosæ ad Oss.
7. ℞ Sodæ biborat. ʒss.
   Liq. hydrarg. perchlor. ʒii.
   Aq. rosæ ad ℥iv.

   Useful lotions in acne punctata.
8. Indolent indurated nodules may be touched with acid nitrate of mercury.
9. ℞ Conf. sulphuris ʒi. Every morning. Useful internally.
10. ℞ Ferri arsen. gr. $\frac{1}{6}$
    Pil. ferri carb. gr. iv.
    ft. pil. i.

    Three times a day after meals.

Ague.

1. ℞ Pulv. rhei
   Pulv. jalapæ āā gr. vi.
   Hydrarg. subchlor.
   Quininæ sulph. āā gr. iv.

   A useful powder at first onset.
2. ℞ Quininæ sulph. gr. iii.-x.
   Acid. hydrobromici ʒss.-i.
   Aq. et syr. ad ℥i. ter die.
3. ℞ Quininæ sulph. gr. x.
   Acid. sulph. dil. ♏x.
   Aq. destillat. ♏c.

   A useful hypodermic solution.

4. ℞ Pil. ferri carb. gr. iii.
Acidi arseniosi gr. $\frac{1}{20}$
ft. pil i.
To be taken thrice daily.

5. ℞ Quininæ sulph. gr. ii.
Ferri sulph. exsic. gr. i.
Acidi arseniosi gr. $\frac{1}{20}$
ft. pil. i.
To be taken thrice daily.

6. Warburg's tincture.
Give ℥ss. undiluted, after opening the bowels. Repeat in 3 hours.

7. Chloroformi, ʒss.-i. In milk.
Taken at time of chill, may abort it.

8. An emetic of ipecacuanha taken in the morning sometimes enhances effect of quinine.

9. ℞ Ammonii iodid. ʒi.
Liq. arsenicalis ʒss.
Tr. calumb. ℥ss.
Aq. ℥iss. M.
One teaspoonful three times a day. In malarial enlargement of spleen.

Alcoholism.

1. ℞ Ammon. carb. ʒi.
Table vinegar ℥i.
A useful reviver from drunkenness.

2. ℞ Liq. arsenicalis ♏i.
Tr. capsici ♏i.
Aquam ad ʒi.
Every hour. Useful in persistent vomiting after a debauch.

3. ℞ Camphor. monobrom. gr. v.
Ext. rosæ caninæ q.s.
ft. pil. i.
One or two as required.

℞ Tr. opii ♏x.
Vin. antimon. ♏xx.
Aquam ad ℥ss.
Every hour as required.

℞ Chloral hydrat. ʒii.
Potass. bromid. ʒii.
Syrup. tolut. ℥i.
Aq. chloroformi ℥iii.
Aquam ad ℥vi.
℥ss.-℥i. every 2 or three hours. Useful sedatives in acute alcoholism. Use with caution.

4. ℞ Potass. bromid. ʒiss.
Tr. digitalis ʒi.
Tr. capsici ʒss.
Aquam camph. ad ℥vi.
℥i. thrice daily.

℞ Potass. bromid. ʒiss.
Inf. quassiæ ℥vi.
℥i. thrice daily.

℞ Zinci oxidi gr. iii.
Ext. gentianæ q.s.
ft. pil. i. Thrice daily.

Anæmia.

1. All forms of iron.

2. ℞ Ferri sulph. gr. ii.
Magnes. sulph. gr. x.-xx.
Acidi sulph. dil. ♏x.
Aq. menth. pip. ℥i.
Thrice daily.

℞ Pil. aloes et myrrh. gr. iv.
Ext. nucis vom. gr. $\frac{1}{2}$
Ext. belladonnæ gr. $\frac{1}{4}$
ft. pil. i. daily after dinner, in constipation with anæmia.

3. ℞ Potass. bromid. ʒi.
Ferri et am. cit. ʒss.
Sp. ammon. ar. ʒi.
Aquam ad ℥vi.
℥i. thrice daily. In anæmia with nervousness.

4. ℞ Liq. arsen. hydroch. ♏iii.-v.
Tr. ferri perch. ♏v.
Aquam ad ℥ss.
Thrice daily in certain cases of anæmia—diluted.

5. ℞ Easton's syrup ʒss.-i.
Thrice daily in water after meals.

6. ℞ Calcii phosph. gr. ii.
In milk 3 or 4 times daily. In anæmia of rapidly growing children.

7. Oleum morrhuæ.

Angina Pectoris.

1. ℞ Sp. ætheris co. ʒi.
Liq. opii sed. ♏x.
Aq. camph. ad ℥ss.
Occasionally in water as required.

2. ℞ Liq. arsenicalis ♏iii.-vi.
Aq. ad ℥i.
Thrice daily for cardiac pain.

3. ℞ Quininæ sulph. gr. i.
Acid. arseniosi gr. $\frac{1}{30}$
Glycer. tragac. q.s.
ft. pil. i.
From 2 to 4 a day in chronic angina.

4. ℞ Nitro-glycerine ♏i.
Sp. vini rect. ♏c.
One to six drops on sugar every four hours.

5. Nitrite of amyl.
Four or five drops to be inhaled from an handkerchief during paroxysm.

Apoplexy.

1. *Croton oil* ♏i.-ii., with a little butter, and placed on the back of the tongue. A useful purgative.

Ascarides.

1. Santonin gr. ii.-iv.
Early morning before food. To be followed by a dose of castor oil. For round or thread-worms.

2. ℞ Aquæ calcis.
or ℞ Sodii chlorid. ʒi. ad aq. Oi.
or ℞ Tr. ferri perch. ʒi. ad aq. Oi.
or ℞ Infusi quassiæ.
As enemata for thread-worms.

3. ℞ Potass. bromid. gr. iv.-x.
Decoc. aloes co. ʒii.-iv.
Twice or thrice daily. Useful both for round or thread-worms.

4. ℞ Pulv. scammon. co. gr. v.-x.
Pulv. aromat. gr. v.
A useful purgative in cases of thread-worms.

5. ℞ Boracis ʒi.
Liq. hydrarg. perchlor. ℥i.
Aq. ad ℥vi.
Useful local application to anus, for itching.

Ascites.

1. ℞ Elaterini gr. $\frac{1}{12}$
Pulv. scillæ
Pulv. digitalis āā gr. i.
Ext. hyoscyami gr. iss.
ft. pil. i. To be taken every other night.

2. ℞ Copaibæ resinæ gr. x.-xv.
Ætheris q.s.
Mist. amygdalæ ℥i.
Thrice daily. Not admissible with albuminuria.

Asthma.

1. ℞ Chloral hydrat. gr. xx.
Syrupi tolut. ʒii.
Aquæ chloroformi ℥ss.
Aquam ad ℥i.
In water every two hours during the paroxysm, until under influence.

2. ℞ Potass. iodid. ʒii.
Liq. morph. hyd. ʒiss.
Tr. scillæ
Tr. lobeliæ
Syrupi. āā ℥i.
One tea-spoonful in water thrice daily in asthma with emphysema and bronchitis.

3. ℞ Liq. arsenicalis ♏iv.
Aquam ad ℥i.
Useful when given for long period in nervous asthma.

4. *Hypodermic injections* of morphia and atropia.

5. Strong hot *coffee* without sugar or milk, given on an empty stomach.

6. *Inhalation* of smoke from tobacco, or stramonium cigarettes.

7. ℞ Potass. iodid. gr. v.-x.
Vini colchici ♏xv.
Aq. camphoræ ℥i.
Thrice daily in gouty bronchitic asthma.

8. *Inhalation* of the spray of ipecacuanha wine, in bronchitic asthma.

9. *Inhalation* of the smoke of burning nitre paper.

10. ℞ Potass. nitrat. ℥ss.
Pulv. anisi fruct. ℥ss.
Pulv. stramonii fol. ℥i.
Ignite and inhale smoke.

11. Chloral hydrate gr. xv.-xx.
Every four hours, until paroxysm subsides.

Bed-sores.

1. Application of *alcohol* to the skin, in threatening bed-sore.

2. *Glycerine* rubbed into parts exposed to pressure, a good preventive of bed-sores.

℞ Argenti nitrat. gr. v.
Sp. æth. nitrosi ℥i.
To be painted on skin, in threatening bed-sore.

4. *Iodoform*, dusted over open sore.

5. Ung. resinæ or balsam peruv.
Useful dressing for open bed-sore.

Biliousness.

1. ℞ Res. podophylli gr. $\frac{1}{4}$-$\frac{1}{2}$
Pulv. capsici gr. $\frac{1}{4}$
Pulv. rhei gr. ii.
Ext. belladon. gr. $\frac{1}{2}$
ft. pil. sec. art.
℞ Res. podophylli gr. $\frac{1}{4}$
Ext. coloc. co. gr. ii.
ft. pil. i.
℞ Pil. hydrarg.
Ext. coloc. co. āā gr. ii.
ft. pil. i.
One or two of either of the above pills at bed-time, followed by a saline aperient draught in the morning.

2. ℞ Magnes. carb. ʒii.
Pulv. rhei ʒi.
Vini ipecac. ʒii.
Pulv. aromat. ℈ii.
Aq. menth. pip. ad ℥viii.
℥i.-iss. every morning.

3. Ipecacuanha in small and long continued doses.

4. ℞ Ext. nucis vom. gr. $\frac{1}{3}$
Ext. coloc. co. gr. iss.
Ext. hyoscyam. gr. iss.
ft. pil. i.
Twice daily. In chronic hepatic sluggishness.

5. ℞ Ammon. chlorid. gr. v.-xv.
Aquæ ℥ss.
Twice or thrice daily in milk. Very useful in biliousness with hepatic congestion.

Boils.

1. *Collodion* to be painted over boil at very first appearance.

2. ℞ Ext. belladonnæ ʒii.-iv.
Glycerini ℥i.
A useful application. Reduces inflammation and allays pain.

3. ℞ Liquoris potassæ ♏xv.-xx.
Infusi cinchonæ ℥i.
Thrice daily in water.

4. A tea-spoonful of German yeast on an empty stomach thrice daily may be tried in obstinate cases.

5. ℞ Calcii sulphid. gr. $\frac{1}{10}$-$\frac{1}{2}$
Sacch. albi q.s.
Every 2 or 3 hours. Valuable in chronic cases.

6. *Tonics* of which the best are quinine, and mineral acids with bark.

Bright's disease.

1. *Tr. Aconiti*—in doses of ♏i. every hour on first appearance of nephritis after scarlet fever.

2. ℞ Potassii tart. acid. ℈i.
Vini ipecac.
Tr. digitalis āā ♏v.
Sp. ætheris nit. ♏v.
Aquam ad ℥i.
Three or four times daily. In post-scarlatinal nephritis.

3. *Tr. cannabis indicæ*—in doses of ♏v.-x. three or four times daily. Said to be useful as diuretic in cases with bloody urine.

4. *Pulv. jalapæ co.*—the most useful purgative in Bright's disease. The addition of elaterium (gr. $\frac{1}{12}$) greatly increases the effect.

5. ℞ Tr. ferri perchlorid.
Tr. nucis vom. āā ♏x.
Sp. æth. nitrosi ʒss.
Aquam ad ℥i.
Thrice daily. A useful formula.

6. ℞ Potassii tart. acid. ʒss.
Tr. digitalis ♏x.
Sp. æth. nit. ♏xx.
Decoct. scoparii ad ℥i.-ii.
Thrice daily. A useful diuretic.

7. Camphorated vapour baths. Half an ounce of camphor for each bath.

Bronchitis.

ACUTE.

1. ℞ Vin. antimon. ♏x.-xv.
Liq. ammon. acet. ʒi.-ii.
Syrupi tolutani ʒss.
Aquam ad ℥i.
Thrice daily. Relaxing expectorant in early stages.

2. ℞ Vin. ipecac. ♏v.
Tr. camph. co. ♏x.
Liq. ammon. acet. ♏xx.
Syrup. et aq. ad ℥ss.
Every three or four hours. A useful expectorant for children.

3. ℞ Ammon. carb. gr. ii.-v.
Ammon. chlorid. gr. v.
Sp. chloroformi ♏x.
Syr. tolutani ʒss.
Aquam ad ℥i.
Thrice daily. Useful stimulant expectorant in later stages.

4. ℞ Chloral hydrat. gr. v.
Oxymel scillæ
Syrupi tolutani āā ʒss.
Occasionally when cough is troublesome.

5. ℞ Quin. sulph. gr. i.
Acid. phosph. dil. ♏xv.
Syrup. tolutani ♏xx.
Aquam ad ℥i.
Thrice daily. Useful tonic in advanced stage.

6. ℞ Potass. iodid.
Ammon. carb. āā gr. iv.
Vin. colchici ♏x.
Tr. scillæ
Tr. hyoscyami āā ♏xv.
Aq. camph. ad ℥iss.
Thrice daily. In acute gouty bronchitis.

7. ℞ Ol. terebinth. ♏xx.-xxx.
Mist. amygdalæ ℥i.
Every 4 or 6 hours. In suffocative bronchitis.

CHRONIC.

1. ℞ Potass. nitrat. gr. iii.
Tr. conii
Sp. æth. nit.
Oxymel scillæ āā ♏xx.
Infusum senegæ ad ℥i.
Thrice daily.

℞ Ammon. chlorid. gr. x.
Sp. ætheris co. ♏x.
Ext. glycyrrhizæ ♏x.
Inf. senegæ ad ℥i.
Thrice daily. Useful stimulating expectorants, especially in bronchitis of the aged.

2. ℞ Picis liquidæ gr. ii.
Pulv. aromat. q.s.
ft. pil. i.
Thrice daily. In paroxysmal cough with fœtid expectoration and bronchial dilatation.

3. ℞ Tr. ferri perchlor. ♏xii.
Ætheris ♏xv.
Aquam ad ℥i.
Thrice daily.

℞ Quin. sulph. gr. i.
Acid. hydrobromici ʒss.
Syrup. tolut. ʒi.
Aquam ad ℥i.
Thrice daily. Useful tonics in chronic bronchitis.

4. *Inhalations* of compound tinct. of benzoin (ʒi.) or creasote (♏x.-xx.) added to boiling water, lessen secretion.

5. *Sulphur* gr. x. taken in milk thrice daily, useful in abundant and fœtid secretion.

Catarrh.

1. *Tr. aconiti* in ♏i. doses every hour in early stages.

2. ℞ Ammonii carb. gr. v.
Liq. morphinæ hyd. ♏v.
Mist. amygdalæ ʒi.
Every four hours, from the outset of catarrh. A colocynth pill to be taken on the second night.

3. *Quinine* in a single dose of gr. x. is said to abort a catarrh if taken early.

4. ℞ Morph. sulphatis gr. i.
Bismuth. subnit. ʒiii.
Pulv. acaciæ ʒi.
To be used as a snuff at the beginning of a cold.

5. ℞ Camphoræ ʒv.
Ætheris q.s.
Dissolve to the consistence of cream and add—
Ammonii carb. ʒiii.
Pulv. opii ʒi.
Keep tightly corked, gr. iii.-x. to be taken in a little water at bedtime when a cold threatens.

6. ℞ Liq. morphinæ hyd. ♏x.
Vin. antimon. ♏x.
Aquam ad ℥i.
Repeated twice at intervals of three hours said to check catarrh on first onset.

Chlorosis.

1. ℞ Ferri sulph. exsic. gr. v.
Glycer. tragacanth. q.s.
ft. pil. i.
Two to six or eight daily.

Cholera and Choleraic Diarrhœa.

1. *Castor oil* in dose of ℥ss., followed in an hour or so by a table-spoonful of brandy with ♏v.-x. of laudanum.

2. ℞ Pulv. opii gr. i.
Piperis gr. ii.
Assafœtidæ gr. iii.
ft. pil. i. sec. art.
To be repeated occasionally as required.

3. ℞ Sp. camphoræ ♏vi.
Every ten minutes in water till symptoms abate. Useful in summer diarrhœa.

4. *Chloroform* in doses of ♏v.-x. every hour, said to relieve vomiting and cramps.

or ℞ Chloroformi ♏x.
Tr. opii ♏v.-x.
Sp. vini rect. ʒi.
Aquam ad ℥i.
A formula recommended.

5. ℞ Plumbi acetat. gr. xxiv.
Liq. morph. hyd. ʒi.
Acid. acet. dil. ♏xii.
Aq. destillat. ad ℥vi.
℥ss. every two hours in water.

6. ℞ Liq. hydrarg. bichlor. ♏x.
Aquam ad ʒi.
Every hour.

or ℞ Hydrarg. cum cretâ gr. $\frac{1}{6}$
Sacchari q.s.
Every hour. Useful in choleraic diarrhœa.

Chorea.

1. *Liq. arsenicalis* in gradually increasing doses, with or without iron.

2. *Sulphate of zinc* in gradually increasing doses, beginning with gr. ii. thrice daily.

3. ℞ Strychninæ sulph. gr. ii.
Aquæ ℥i.
Five drops thrice daily for a child of ten years.

4. Chronic cases do best with cod-liver oil and ferruginous tonics.

Colic.

1. Hot fomentations, and warm baths.

2. ℞ Magnes. carb. ʒiss.
Ammon. carb. ʒss.
Tr. lavand. co. ʒii.
Aq. menth. pip. ad ℥iv.
A table-spoonful occasionally as required. In simple colic.

3. ℞ Sp. ætheris ♏xx.
Liq. morph. hyd. ♏v.
Aq. menth. pip. ad ℥i.
Occasionally as required. In simple colic.

4. ℞ Sodii sulpho-carbolat. gr.xv.
Tr. belladonnæ
Sp. chloroformi āā ♏x.
Aquam ad ℥i.
Thrice daily in simple flatulent colic.

5. ℞ Creasoti ♏i.
Pulv. aromat. gr. iv.
Mucilaginis q.s.
ft. pil. i.
Three or four daily in simple colic.

6. ℞ Ol. ricini ℥i.
Tr. opii ♏xx.
A useful draught in lead colic.

7. ℞ Ext. opii gr. ½-i.
Ext. belladon. gr. ¼
Ext. hyoscyami gr. ii.
ft. pil. i.
Every four or six hours as required. In colic with intestinal obstruction.

8. *Enemata* of turpentine or castor oil are useful in colic with simple constipation.

Congestion.

CEREBRAL CONGESTION.

1. ℞ Potass. bromid. gr. xv.
Ext. ergot. liq. ♏xx.-xxx.
Aquam ad ℥i.
Thrice daily. In asthenic congestion.

2. ℞ Zinci phosphidi gr. iii.
Ext. nucis vom. gr. x.
ft. pil. 30.
One thrice daily. In asthenic congestion.

3. ℞ Liq. arsenicalis ♏iii.
Tr. ferri perchlor. ♏v.
Aquam ad ℥i.
Thrice daily. In congestion due to mental strain.

4. Alcohol of service in some cases of *passive* congestion.

HEPATIC CONGESTION.

1. Saline aperients.

2. ℞ Sodii sulphat. ʒii.
Succi taraxaci ʒi.
Aquam ad ℥ii.
A useful morning draught.

3. The mineral waters of Carlsbad, Friedrichshall, or Hunyadi Janos.

4. ℞ Ammonii chlordi. gr. x.-xv.
Aquæ ℥i.
Three times a day, in milk.

5. ℞ Acid. nitro-mur. dil. ℥i.
Aquam ad Oi.
Compress to be soaked in this fluid, and applied over the liver.

CONGESTION OF LUNG (see PNEUMONIA).

Constipation.

1. ℞ Ext. aloes gr. i.
Ext. nucis vom. gr. ½
Ext. gentian. gr. iii.
ft. pil. i.
A useful dinner pill in chronic constipation.

2. Decoction of aloes is a valuable aperient for females with pelvic troubles.

3. ℞ Ext. belladon. gr. ¼
Ext. aloes gr. i.
Ext. gentian. gr. ii.
ft. pil.
Once or twice daily in atonic constipation with dyspepsia.

4. *Croton oil* ♏i.-ii. To be given in a little butter and placed on the back of the tongue. A very powerful purgative.

5. *Ipecacuanha* in dose of gr. i. every morning said to be useful in chronic constipation with slow digestion.

6. ℞ Mist. sennæ co. ℥i.-iss.
or ℞ Magnes. sulphat. ʒii.
Magnes. carb. gr. xx.
Aq. menth. pip. ad ℥iss.

or ℞ Sodii sulphatis ʒi.-ii.
Taken in a tumbler of water in the morning.
or ℞ Seidlitz powder.
Useful saline aperients.

7. ℞ Pil. coloc. co.
Pil. hydrargyri ââ gr. iiss.
ft. pil. i.
or ℞ Pil. coloc. et hyoscyam. gr. v. (B.P.)
or ℞ Pil. rhei co.
Pil. hydrargyri āā gr. iss.
ft. pil. i.
or ℞ Podophyllin gr. ¼-½
Pulv. ipecac. gr. ½
Ext. gentianæ gr. iii.
ft. pil. i.
or ℞ Podophyllin gr. ¼-½
Pil. rhei co. gr. ii.
Ext. hyoscyam. gr. ii.
ft. pil. i.
Useful aperient pills.

8. Castor oil, confection of sulphur, confection of senna, manna, and tamarinds are mild laxatives.

9. Pulv. jalapæ co. (gr. xx. - ʒi.) with or without the addition of gamboge (gr. i.-ii), elaterium (gr. ¼-½), and bitartrate of potash, are useful hydragogue aperients.

10. Pulv. scammon. co. (gr. x.-xx.) with or without the addition of calomel is a valuable aperient for dislodging intestinal worms.

11. Enemata—which may consist of simple soap and water, or contain turpentine or castor oil suspended in thin gruel.

Convulsions.

1. Warm baths.
2. Inhalation of chloroform.
3. ℞ Potass. bromid. gr. iii.
Chloral hyd. gr. i.
Aq. et syrup. ad ʒi.
Every 4 or 6 hours in convulsions of young children.
4. Morphia injected subcutaneously will often stop puerperal convulsions.

Croup.

1. Aconite—drop doses of the tincture every half hour useful in early stages.
or ℞ Tr. aconiti ♏ii.
Vin. ipecac. ♏iv.
Sp. æth. nit. ♏x.
Aq. et syr. ad ʒi.
Every three hours.

2. ℞ Potass. chlorat. gr. iv.
Ammon. chlorid. gr. ii.
Aq. et syr. ad ʒii.
Every 20 or 30 minutes in mild cases.

3. ℞ Potass. chlorat. gr. v.
Tr. ferri perchlor. ♏iii.
Aq. et syrup. ad ʒii.
Every three or four hours.

4. An emetic followed by
℞ Potass. iodid. gr. ii.
Sp. ammon. ar. ♏v.
Syr. et inf. senegæ ad ʒii.
Every three hours.

5. ℞ Alum ʒi.
or Zinci sulph. gr. xv.-xx.
or Cupri sulph. gr. ij.-iv.
Useful emetics. To be given in honey or syrup every 15 minutes, till vomiting.

6. Lime water.
To inhale spray from atomiser. Useful also to keep tubes clean after tracheotomy.

Diarrhœa.

1. ℞ Olei ricini ♏xxx.
Sp. chloroformi ʒi.
Liq. morph. hyd. ʒi.
Pulv. acaciæ ʒi.
Aq. et syr. ad ℥iv.
One or two teaspoonfuls every one or two hours, as required. Valuable for general use.

2. ℞ Bismuthi subnit. ℈i.
Acid. tannic.
Pulv. ipecac. co. āā gr. iii.
Thrice daily. In dysenteric diarrhœa of adults.

℞ Bismuthi subnit. gr. ii.
Pulv. ipecac. co.
Pulv. cinnam. co. āā gr. ½.
Every 3 or 4 hours. In dysenteric diarrhœa of children.

3. Liq. arsenicalis in drop doses every 3 or 4 hours useful in lienteric diarrhœa.
4. ℞ Tr. opii
Tr. capsici āā ♏i.
Sp. chloroformi ♏ii.
Aquam ad ʒi.
Occasionally as required. In diarrhœa persisting after expulsion of exciting irritant.
5. ℞ Acid. sulph. arom. ♏xv.
(Tr. opii ♏v.)
Aq. menth. pip. et syr. ad ℥i.
Every 4 hours. In summer diarrhœa of adults.
℞ Acid. sulph. dil. ♏iii.
Syr. rheados ♏xx.
Aquam ad ʒi.
Every 4 hours. In simple summer diarrhœa of children.
6. ℞ Vini ipecac. ♏i.
Aquam ad ʒi.
In diarrhœa of children with slimy stools and obstinate vomiting.
7. ℞ Plumbi acetat. gr. ii.
Pulv. opii gr. ¼-½
Ext. hyoscyam. gr. iii.
ft. pil. i.
As required. Valuable in obstinate cases.
8. ℞ Liq. hydrarg. perchlor. ♏xii.
Aquam ad ʒi.
Every hour. In dysenteric diarrhœa with slimy bloody stools and tenesmus (children).
or Hydrarg. cum cretâ gr. ⅙.
Every hour. In watery offensive stools with vomiting (children).
9. *Opium* in various forms in dose according to age. Dover's powder very useful.
10. ℞ Zinci oxidi gr. ii.-iv.
(Tr. opii ♏¼-½)
Aq. et mucilagin. ad ʒi.
Every 4 hours in diarrhœa of children.
11. ℞ Argent. nit. gr. i.
Tr. opii ♏x.-xx.
Mucil. ad ℥iss.
ʒi. thrice daily.
or ℞ Liq. ferri pernit. ♏xv.
Acid. nitric. dil. ♏xv.
Syrup. zingib. ℥ss.
Aq. anethi ad ℥iss.
ʒi. every 6 hours.
Useful in chronic diarrhœa of children.

Diphtheria.

1. ℞ Potass. chlorat. gr. iv.
Tr. ferri perchlor. ♏iv.
Aq. et syrup. ad ʒii.
Every two or three hours.
2. ℞ Acidi sulphurosi ʒvi.
Syrupi ℥iv.
Sol. (saturated) potass. chlorat. ad ℥vii.
From ½ to 2 teaspoonfuls every hour.
3. ℞ Quin. sulph. gr. ½
Tr. ferri perch. ♏iii.
Potass. chlorat. gr. iii.
Aq. et syr. ad ℥ss.
Every 2 or 3 hours in asthenic cases.
4. Local applications to throat.
℞ Acidi hydrochlor.
Aquæ æq. partes.
For a *single* application only.
℞ Acid. salicylic. ʒss. *or*
Acid. carbol. ʒss.
Glycerini ℥ii.
Aquæ calcis ℥viii.
As an application or spray.
℞ Tr. ferri perchlor. ʒiss.
Glycerin. et aq. ad ℥ii.
5. Gradually increased doses of strychnia together with galvanism required in diphtheritic paralysis.

Dropsies.

1. Saline purgatives in a minimum quantity of water.

2. ℞ Elaterii gr. ¼
Pulv. scillæ
Pulv. digitalis āā gr. i.
Ext. hyoscyami gr. iss.
ft. pil. i.
Every other night.

3. ℞ Pulv. scillæ gr. iss.
Pulv. digitalis gr. ss.
Pil. hydrarg. gr. ii.
ft. pil. i.
Twice or thrice daily, followed by
℞ Potass. acetat. gr. xx.
Sp. æth. nit. ʒss.
Decoct. scoparii ℥iss.

4. ℞ Sodii sulphat.
Potass. bitart. āā ℥i.
Sp. æth. nit. ℥i.
Aquam ad ℥vii.
℥ss. thrice daily in a tumbler of barley water.

5. ℞ Potass. nitrat. gr. iii.-v.
Sp. æth. nit. ʒss.
Infusi digitalis ʒi.-ii.
Aquam ad ℥i.
Thrice daily.

6. Mist. ferri et arsenicalis ℥i.
Thrice daily in anæmic dropsy.

7. ℞ Liq. ammon. acet. ℥i.
Acid. acetic. dil. ʒiiss.
Tr. ferri perch. ʒii.
Aquam ad ℥viii.
℥i. thrice daily in chronic renal dropsy.

Dysentery.

1. Pulv. ipecac. gr. xx.-xxx. To be given in as small a quantity of fluid as possible. To be repeated in 8 or 10 hours if necessary. Give gr. x. at bedtime for 2 or 3 nights after.

2. ℞ Olei terebinth.
Tr. opii āā ʒiii.
Acaciæ pulv.
Sacchari alb. āā ʒiii.
Aq. menth. pip. ℥iii.
One teaspoonful every four hours, after the bowel has been cleared by a dose of castor oil.

3. ℞ Liq. hydrarg. perch. ♏xii.
Aquam ad ʒi.
Every hour.

4. ℞ Tr. benzoin. co. ʒss.
Tr. catechu ʒi.
Tr. opii ♏x.
Decoct. hæmatoxyli ad ℥i.
Thrice daily. Recommended in chronic dysentery.

5. ℞ Liq. ferri pernitrat. ♏xv.-xx.
Sp. chloroformi ♏x.
Aquam ad ℥i.
Thrice daily in chronic dysentery.

6. ℞ Cupri sulphat. gr. x.-xx.
Aquæ Oi.
As an enema in chronic dysentery.

Dyspepsia.

1. *Alkalies* with vegetable bitters very useful in various forms of dyspepsia. The addition of liq. potass. (♏x.) is useful in some forms of painful digestion.
℞ Sodii bicarb. gr. xv.-xx.
Tr. hyoscyam. ♏xv.
Inf. calumb. *vel* quassiæ ℥i.
Useful in dyspeptic vertigo.

2. The *mineral acids* with vegetable bitters useful in various forms of dyspepsia. In acid dyspepsia should be taken *before* food.

3. ℞ Sodii sulphitis ʒi. *vel*
Sodii sulpho-carbol. ʒi.
Tr. nucis vom. ʒiii.
Aquam ad ℥iv.
ʒi.-ii. thrice daily after food in flatulent dyspepsia.

4. ℞ Ammon. carb. gr. v.
Potass. bicarb. gr. x.
Inf. aurantii ad ℥i.
For loss of appetite with flatulence.

5. *Liq. arsenicalis* in drop doses useful in neuralgic gastrodynia. Also in morning vomiting of drunkards.

6. *Bismuth* mixtures are useful in painful digestion. The addition of hydrocyanic acid is often desirable.

7. ℞ Pulv. rhei ʒi.
Sodii bicarb. ʒiss.
Aquæ menth. pip. ℥iv.
℥ss. before meals in dyspepsia with constipation.

8. ℞ Ext. aloes.
Ext. gentian. āā gr. ii.
Ext belladon.
Ext. nucis vom. āā gr. ¼
ft. pil. i.
A useful dinner pill.

9. ℞ Quininæ sulphatis
Pulv. ipecac. āā gr. i.
Ext. gentian. gr. ii.
ft. pil. i.
An excellent dinner pill in slow digestion.

10. *Ferruginous tonics* generally are useful in atonic dyspepsia.

11. *Nux vomica* is a valuable tonic in weak digestion.

12. ℞ Bismuthi subnit.
Carbonis ligni āā gr. v.
In extreme flatulence.

Eczema.

Acute Eczema.

1. *Alkaline bath.*—6 oz. bicarbonate of potash or soda to 30 gallons of water.

2. *Milk and water* or *weak gruel* are useful washes.

3. ℞ Zinci oxidi
Pulv. amyli æq. partes.
A good absorbent powder. If much burning, add *camphor*—ʒss. to 1 oz. of powder.

4. ℞ Lotio plumbi acetatis.

5. ℞ Liq. plumbi acetat. ʒiss.
Vini opii ʒi.
Ung. zinci *vel* sambuci ℥i.
ft. ung.
When the skin is burning and irritable.

6. ℞ Calaminæ ʒi.
Liq. plumbi acetat. ♏x.
Acid. hydrocyan. dil. ♏xx.
Glycerini ♏xx.
Adipis ℥i.
ft. ung.

7. ℞ Glycerini boracis ℥i.
Aquæ rosæ ℥ii.
℞ Boracis ℥iss.
Glycerini ʒi.
Adipis ℥i.
ft. ung.

8. ℞ Potass. cyanidi gr. vi.
Ung. cetacei. (cold cream) ℥i.
ft. ung.
To relieve itching.

9. ℞ Camphoræ ʒss.
Sp. vini rect. q.s.
Ung. zinci ℥i.
ft. ung.
Soothing ointment.

Chronic Eczema.

1. *Arsenic*—in chronic eczema with scaliness.

2. ℞ Picis liquid. ♏i.-ii.
Pulv. aromat. q.s.
ft. pil. i.
Thrice daily, in some forms.

3. ℞ Hydrarg. ammon. *vel*
Hydrarg. oxid. rubri gr. v.
Ung. simplicis ℥i.
ft. ung
A valuable ointment in subacute and chronic eczema.

4. ℞ Lotio acid. carbolic. 1 in 40.
or ℞ Acidi carbolici ʒi.
Ung. zinci ℥i.
ft. ung.
To relieve itching.

5. ℞ Picis liq. *vel* ol. cadini
Sp. vini rect.
Saponis mollis æq. partes.
Valuable application in some forms of chronic local eczema.

6. ℞ Glycerini acid. tannic.
Useful in eczema of the ears and other parts.

Emphysema.

1. ℞ Tr. ferri acetat. ♏xv.
Sp. chloroformi ♏xx.
Aquam ad ℥i.
Thrice daily.

2. ℞ Tr. lobel. æth. ʒii.
Sp. æth. sulph. ʒiii.
Tr. conii ʒii.
Mist. amygdalæ ad ℥vi.
A table-spoonful every three hours in the paroxysmal cough of emphysema.

3. ℞ Potass. iodid. ℈ii.
Vin. ipecac. ʒiss.
Tr. scillæ ʒi.
Aq. et syr. ad ℥viii.
A table-spoonful thrice daily.

4. Cod-liver oil.

5. Arsenic—systematic course.

6. ℞ Ammon. carb. gr. iv.
Vini ipecac. ♏x.
Sp. chloroformi ♏x.
Inf. gentian. ad ℥i.
Thrice daily in emphysema with bronchitis.

Epilepsy.

1. Bromide of potassium in doses of ten to twenty grains thrice daily.

2. ℞ Potass. iodid. gr. v.
Potass. bromid. gr. x.
Ammon. bromid. gr. x.
Potass. bicarb. gr. x.
Infus. calumb. ℥i.
Twice or thrice daily half an hour before meals. A useful combination.

3. ℞ Potass. bromid. gr. xv.
Liq. atrop. sulph. ♏ii.
Aquam ad ℥i.
Thrice daily.

4. ℞ Potass. bromid. gr. x.
Liq. arsenicalis ♏iv.
Aquam ad ℥i.
Thrice daily.

5. ℞ Zinci bromid. ʒi.
Syrupi simp. ℥i.
Ten to twenty drops in water thrice daily.

6. ℞ Zinci oxidi gr. ii.-iii.
Ext. glycyrrhizæ q.s.
ft. pil. i.
One or two thrice daily.

7. ℞ Zinci valerianat. gr. iii.
Ext. belladon. gr. ¼
Ext. glycyrrhizæ q.s.
ft. pil. i.
Thrice daily.

8. ℞ Boracis gr. xv.-xx.
Aquæ ℥ss.
Thrice daily. In chronic cases.

Fever.

1. ℞ Vin. antimon. ♏x.
Liq. ammon. acet. ʒi.
Aquam ad ℥i.
Every 4 or 6 hours.
or ℞ Potass. nitrat. gr. v.
Vin. antimon. ♏x.
Liq. ammon. acet. ʒii.
Aq. camphor. ad ℥i.
Every 4 hours.
or Solutions of citrate, chlorate, bitartrate, or acetate of potash, sweetened and flavoured with lemon-juice.
Useful salines in mild fever.

2. ℞ Potass. chloratis ʒiii.
Acid. hydrochlor. dil. ʒii.-iii.
Syrup. zingiber. ℥i.
Decoct. hordei Oii.
or ℞ Acid. phosphor. dil. ʒiii.
Glycerin. ℥i.
Decoct. hordei Oii.
Useful acid drinks in mild fevers.

3. *Tr. Aconiti* — in drop doses every half hour, or hour, very useful in early stages of fever.

4. *Alcohol* — in later stages of fever, with nervous depression, muttering delirium, and compressible pulse.

5. *Quinine*—gr. x.-xx. *antipyrin*—gr. xx.-xxx. and *antifebrin*—gr. v. Best given about bed-time. Valuable antipyretic remedies in many fevers.

6. ℞ Quininæ sulph. gr. v.
Tr. digitalis ♏x.
Acid. phosph. dil. ♏xv.
Aquam ad ℥i.
Every 4 or 6 hours.
A useful febrifuge where the skin is moist.

7. ℞ Sodii salicylat. gr. x.-xx. *vel*
Quin. salicylat. gr. i.-iii.
Aquæ ℥i.
Every 4 or 6 hours.
Useful febrifuge, especially in rheumatic fever.

8. *Cold baths*, wet sheet packs, and sponging, with tepid or cold water, very useful in reducing temperature.

Gall-stones.

During the Paroxysm.

1. *Opium*, or preferably subcutaneous injections of morphia.

2. ℞ Ext. belladon. gr. $\frac{1}{4}$
Ext. gentian. gr. ii.
ft. pil. i.
Every 2 or 3 hours.

3. *Inhalation* of chloroform.

During the Interval.

4. ℞ Ammon. chlorid. gr. x.
Succi taraxaci ʒi.-ii.
Aquam ad ʒi.

5. ℞ Chloroformi.
Ætheris sulph. āā ʒss.
Olei terebinth. ℥i.
Sacchari albi ʒii.
Mucilaginis ℥ii.
A teaspoonful thrice daily.

6. ℞ Sodæ phosphat. ʒi.
Aquæ ℥i.
Thrice daily.

Gastralgia.

1. ℞ Acid. arseniosi gr. $\frac{1}{25}$
Ext. gentian. q.s.
ft. pil. i.
Thrice daily.

2. ℞ Bismuthi carb. gr. v.
Sodii bicarb. gr. x.
Pulv. tragac. co. gr. v.
Acid. hydrocyan. dil. ♏iii.
Aquam ℥i.
Thrice daily.

3. *Charcoal*—in dose of gr. xxx. in gastrodynia with much flatulence.

4. *Pepsine*—in dose of gr. v.-x. with meals, in gastrodynia, following the use of animal food.

Gout.

1. An aperient pill at bed-time followed by a saline aperient draught in the morning, necessary at the outset.
Mercury to be avoided if albuminuria.

2. ℞ Potass. iodid. gr. ii.
Vini colchici ♏xv.
Tr. aconiti ♏iii.
Infus. rhei ad ℥i.
Thrice daily in acute gout.

3. ℞ Potass. iodid. gr. iii.-v.
Vini colchici ♏xv.
Magnes. sulph. gr. xx.-xxx.
Tr. hyoscyami ʒss.
Aquam menth. pip. ad ℥i.
Thrice daily in acute gout with constipation.

4. ℞ Hydrarg. subchlor.
Ext. aloes
Ext. colchici acet.
Pulv. ipecac. āā gr. i.
ft. pil. i.
Thrice daily in gout with hepatic congestion.

5. The bicarbonate, citrate, or acetate of potash with or without colchicum, and some vegetable bitter infusion is often of service in sub-acute gout.
or ℞ Lithii carb. gr. iii.-vi.
Aquæ ℥iii.
As a draught, twice a day.

6. ℞ Potass. bromid. gr. xx.
Tr. cannab. ind. ♏xv.
Tr. lupuli ʒi.
Aquam camph. ad ℥i.
A good draught at bed-time for sleeplessness.
Opium is contra-indicated.

7. ℞ Potass. iodid. ʒi.
Potass. bicarb. ℥i.
Aquæ bullientis Oi.
As local external application to the inflamed joint. Laudanum may be added.

8. ℞ Potass. iodid. gr. iv.
Potass. bicarb. gr. xv.
Vini colchici ♏v.
Inf. cinchonæ ℥i.
Thrice daily, in chronic gout.

9. *Iron* and *cod-liver oil* are often of service in chronic atonic gout.

10. *Mineral waters* of Vichy, Wiesbaden, and Carlsbad.

Hæmorrhage.

1. *Cold*—as an external application, or by sucking ice.

2. ℞ Acid. gallic. gr. x.-xv.
Acid. sulph. arom. ♏xv.
Tr. cinnamon. ʒii.
Aquam ad ℥i.
As a draught, every four or six hours.

℞ Acid. gallic. gr. xv.
Pulv. ipecac. co. gr. v.
As a powder, every six or eight hours.

3. ℞ Ext. ergot. liq. ʒss.-ʒi.
Aquam ad ʒiss.
As a draught occasionally as required.

4. ℞ Ferri ammon. sulph. gr.v.-x.
Aquæ ℥i.
Every 6 or 8 hours. An excellent styptic in hæmatemesis.

5. ℞ Ol. terebinth. ♏x.-xx.
Mist. amygdalæ ℥i.
Every 3 or 4 hours. In hæmaturia smaller doses to be used (♏iii.-v.) and with caution.

6. ℞ Infus. matico ℥iss.
Three or four times daily in hæmaturia.

7. *Tr. Hamamelis*—♏ii.-iv. in water every 2 or 3 hours, serviceable in hæmoptysis and hæmatemesis.

Headache.

BILIOUS HEADACHES.

1. ℞ Quin. sulph. gr. ii.
Pulv. rhei gr. iii.
Mucilaginis q.s.
ft. pil. i.
To be taken at bed-time.

2. ℞ Sodii bicarb. gr. xv.
Tr. nucis vom. ♏vi.
Inf. gentian. co. ℥i.
Thrice daily before meals.

3. ℞ Infusi sennæ
Infusi gentian. co. āā ℥ss.
Once or twice daily.

4. ℞ Pil. hydrarg.
Pil. rhei co. āā gr. iv.
Ext. hyoscyam. gr. ii.
ft. pil. ii.
At bed-time.

PLETHORIC HEADACHES.

1. Saline aperients.

2. ℞ Ferri sulph. gr. ii.
Magnesii sulph. gr. xx.
Acid. sulph. dil. ♏xii.
Tr. card. co. ʒi.
Aquam ad ℥i.
Thrice daily.

3. ℞ Tr. ferri perchlor. ♏xv.
Acid. hydrochlor. dil. ♏v.
Sp. chloroform. ♏x.
Aquam ad ℥i.
Thrice daily, especially during climacteric period.

4. ℞ Tr. veratri virid. ♏i.
In water every hour. Useful in congestive headaches recurring at menstrual period.

NERVOUS AND SICK HEADACHES.

1. ℞ Potass. bromid. gr. xx.
Aquæ camph. ℥i.
Thrice daily, in nervous headaches with wakefulness.

2. ℞ Ext. cannab. ind. gr. $\frac{1}{4}$-$\frac{1}{2}$
(Camphor. gr. i.)
Confect. rosæ q.s.
ft. pil. i.
At bed-time, or twice daily. In nervous headaches, with troubled sleep.

3. ℞ *Tr. nucis vom.*—in dose of ♏i. every half-hour or hour in headaches from exhaustion.

4. *Strong tea or coffee* in small quantity for nervous exhaustion. Contra-indicated if it causes palpitation.

5. *Guarana*—ʒss.-i. of the powder. in typical migraine.

6. ℞ Zinci oxidi gr. i.-ii.
Confect. rosæ q.s.
ft. pil. i.
Thrice daily after meals. In nervous headaches.

7. *Phosphorus*—gr. $\frac{1}{50}$-$\frac{1}{30}$ in the form of a pill, thrice daily. In nervous headaches.

NEURALGIC HEADACHES.

1. ℞ Quininæ sulph.
Camphoræ
Ext. hyoscyami āā gr. i.
Confect. rosæ q.s.
ft. pil. i.
A very useful pill: to be taken thrice daily.

2. ℞ Butyl-chloral. hyd. gr. v.-x.
Three or four times daily.

3. ℞ Liq. arsenici hyd. ♏v.
Tr. ferri perch. ♏v.
Aquam ad ℥i.
Thrice daily, after meals.

4. ℞ Ung. aconitinæ.
or ℞ Ung. veratrinæ.
Useful local applications.

ORGANIC HEADACHES

1. ℞ Potass. iodid. gr. v.
Tr. cannab. ind. ♏x.
Inf. calumb. ℥i.
Thrice daily, in syphilitic headache.

2. Bromide of potassium (*vide supra*).

3. ℞ Tr. gelsemii ♏xv.-xx.
Aquæ camph. ℥ss.
Every 4 hours, as required.

Heart Disease.

1. ℞ Tr. digitalis ♏x.
Sp. æth. nit. ʒss.
Infusum buchu ad ℥i.
Thrice daily, in simple cardiac debility.

2. ℞ Tr. digitalis ♏x.
Ferri et am. cit. gr. v.
Ammon. carb. gr. v.
Aquam ad ℥i.

Thrice daily. As a cardiac tonic, with palpitation, anæmia, and debility.

3. ℞ Pulv. digitalis, gr. $\frac{1}{2}$-i.
Ferri sulph. exsic. gr. $\frac{1}{4}$
Pulv. capsici gr. i.
Ext. aloes gr. ii.
ft. pil. i.

Twice daily. As a cardiac tonic, with flatulent dyspepsia, and constipation.

4. ℞ Potass. iodid. gr. v.
Potass. bicarb. gr. xv.
Infus. buchu ℥iss.

Thrice daily with a tumblerful of water, in cardiac irregularity, with gouty tendency.

5. ℞ Potass. iodid. gr. v.
Potass. bromid. gr. xv.
Aquæ camph. ℥i.

A useful draught at bed-time, in cardiac dyspnœa.

6. ℞ Pulv. digitalis gr. $\frac{1}{2}$-i.
Ext. belladon. gr. $\frac{1}{2}$
Ferri sulph. exsic. gr. ii.
Syrupi simp. q.s.
ft. pil. i.

Thrice daily, in cardiac dilatation.

7. *Hydragogue Cathartics*, together with digitalis in some form, are indicated in cardiac dilatation with dropsy. Elaterium should always be used with caution.

8. ℞ Tr. ferri perchlor. ♏x.
Tr. digitalis ♏x.-xv.
Sp. æth. nitrosi ♏xx.-xxx.
Aquam ad ℥i.

Thrice daily. A useful general formula in heart disease.

9. *Strychnia*—alone or in combination with iron and digitalis, is a useful tonic, especially in fatty degeneration.

10. ℞ Potassii bromid. gr. x.
Tr. belladon. *vel*
Tr. digitalis ♏x.
Aquæ camph. ℥i.
Thrice daily in palpitation.

11. *Morphia* — hypodermically injected is useful in paroxysmal attacks of palpitation with dyspnœa. Should be used cautiously.

12. ℞ Potass. acetatis ℈i.
Tr. ferri acetat. ♏x.
Sp. æth. nitrosi ʒss.
Tr. digitalis ♏x.
Aquam ad ℥i.
Thrice daily, in cardiac dropsy.

13. *Liq. arsenicalis*—♏iii.-v., well diluted, thrice daily, for cardiac pain and distress.

14. ℞ Liq. ammoniæ fort. ♏v.
Potass. iodid. gr. ii.
Tr. digitalis ♏v.
Aquam ad ℥ss.
Every 1 or 2 hours. In threatening death from coagulation within the heart.

Hiccup.

1. Sucking *ice*.

2. *Morphia* — hypodermically injected.

3. ℞ Chloral hydrat.
Potass. bromid.
Potass. bicarb. āā ʒi
Aq. menth. pip. ad ℥vi.
One tablespoonful every four hours. Useful in some obstinate cases.

4. *Physostigma*—(gr. $\frac{1}{10}$-$\frac{1}{4}$ of the extract) in some obstinate cases. To be repeated as required.

5. *Pilocarpine*—(gr. $\frac{1}{20}$) hypodermically injected, gave relief in a case which resisted all usual remedies.

Hysteria.

1. ℞ Potass. bromid. gr. xv.-xx.
Ferri et am. cit. gr. iii.
Sp. ammon. arom. ♏xx.
Aquam ad ℥i.
Thrice daily, in hysteria with anæmia.

2. *Phosphorus*—in dose of gr. $\frac{1}{100}$ to $\frac{1}{30}$ with or without the addition of strychnia or nux vomica, is an excellent tonic in hysteria (Cox's formula preferred).

3. ℞ Liq. strychniæ ♏iv., gradually increased.
Tr. card. co. ♏xv.
Aquæ camph. ℥i.
Thrice daily, in hysterical paralysis. Paralysed muscles to be galvanised.

4. ℞ Zinci phosphid. gr. $\frac{1}{6}$-$\frac{1}{3}$
Ext. nucis vom. gr. $\frac{1}{2}$
Pil. aloes cum myrrhâ gr. iii.
Twice or thrice daily, in hysteria with constipation.

Jaundice.

1. A mercurial aperient pill at night, followed by a saline aperient draught in the morning, is beneficial in catarrhal jaundice.

2. ℞ Hydrarg. cum cretâ gr. $\frac{1}{6}$-$\frac{1}{3}$
Sacchari alb. q.s.
Thrice daily, together with
℞ Acid. nitro-mur. dil. ♏v.
Tr. nucis vom. ♏v.
Aquam ad ℥ss.
Thrice daily. Useful in catarrhal jaundice.

3. ℞ Hydrarg. subchlor. gr. $\frac{1}{2}$
Pulv. opii gr. $\frac{1}{3}$
Bismuthi subnit. gr. x.
ft. pulv.
Useful in the obstinate vomiting which sometimes accompanies jaundice with hepatic congestion. To be taken every three hours.

4. *Ammon. chlorid.*—in doses of gr. xx. every 5 or 6 hours in jaundice. Best taken in milk.

5. ℞ Acid. benzoic. gr. iii.-x.
Glycerini q.s.
To form one or more pills. To be taken three or four times daily.
or ℞ Ammon. benzoat. gr. x.-xx.
Syrupi aurantii ʒss.
Aquam ad ℥i.
Thrice daily. Useful formulæ in some forms of congestive jaundice.

Laryngismus Stridulus.

1. *Warm baths*—during paroxysm.
2. *Chloroform*—inhalation during paroxysm.
3. ℞ Potass. bromid. gr. iii.-x.
Syrupi aurantii ʒss.
Aquam ad ʒii.
Thrice daily.
Children bear the bromide very well.
3. *Cod-liver oil* and other *tonics* are useful in chronic cases.

Laryngitis.

1. *Hot fomentation* to throat externally, with inhalations of steam, in acute forms.
2. ℞ Vin. antimon. ♏x.
Vin. ipecac. ♏x.
Potass. acetat. gr. xx.
Sp. æth. nitrosi ʒss.
Potass. nitrat. gr. v.
Aquam ad ℥i.
Every 4 or 6 hours. Useful in acute laryngitis of adults with general febrile disturbance.
3. ℞ Ammon. chlorid. ℈i.
Tr. opii ♏v.
Decocti cinchonæ ℥i.
Twice daily at 11 and 4. In acute laryngitis and commencing "colds."
4. ℞ Tr. benzoin. co. ʒi.
Chloroformi, a few drops.
Added to boiling water. For inhalation. A useful sedative in acute laryngitis.
5. *Tannic acid*—in solution of gr. iii.-x. to one ounce of water.
As a spray for inhalation.
6. Local application to the larynx of:—
Sulphate of Copper,
or Nitrate of Silver,
or Chloride of Zinc,
or Perchloride of Iron,
or Glycerine of Tannin.
7. *Inhalation* of turpentine, creasote, or carbolic acid, in chronic laryngitis.

Lead-poisoning.

1. *Hot fomentations* to abdomen in lead colic.
2. *Morphia*—gr. $\frac{1}{6}$ to $\frac{1}{3}$ hypodermically injected, with or without liq. atropinæ sulph. ♏i.-ii. in lead colic.
3. ℞ Potass. iodid. gr. v.-x.
Inf. calumbæ ℥i.
Thrice daily in chronic cases.
4. ℞ Sodii chlorid. ʒss.
Thrice daily dissolved in water has also been recommended in chronic cases.
5. ℞ Ext. coloc. co. gr. ii.
Ext. opii gr. $\frac{1}{2}$
Ext. belladon. gr. $\frac{1}{6}$
ft. pil. i.
Every 4 or 6 hours, till the bowels are open and the pain relieved, in chronic lead constipation.

Lithiasis.

1. ℞ Potass. citrat. ℈i.
Ammon. carb. gr. ii.
Inf. calumbæ ℥i.
Thrice daily.
2. ℞ Liq. potass. ♏x.-xv.
Tr. hyoscyami ʒss.
Infusi buchu ℥iss.
Thrice daily.

Liver—Diseases of.

1. *Aperients*—salines are the best, as magnes. sulph., sodæ sulph., sod. pot. tart., and purgative mineral waters. An occasional mercurial pill is sometimes of great benefit. Podophyllin not so generally useful.

2. *Alkalies*—in the form of carbonates, or bicarbonates, in combination with vegetable bitters.
3. *Chloride of Ammonium* — in doses of not less than gr. x. thrice daily, is of high value. It may be combined with acids or alkalies.
4. *Mineral Acids*—especially the nitro-muriatic in small doses (♏v.) thrice daily; often of great service.

Locomotor Ataxy.

1. ℞ Antipyrin gr. x.
   Aquæ ℥i.
   Thrice daily. To relieve pains of early stages.
2. ℞ Argenti nitratis gr. ¼-⅓
   Ext. nucis vom. gr. ½
   Confect. rosæ q.s.
   ft. pil. i.
   Thrice daily.
   ℞ Argenti nitratis
   Ext. belladon. āā gr. ¼-⅓
   Ext. gentian. q.s.
   ft. pil. i.
   Thrice daily.
3. ℞ Ext. belladon. gr. ¼
   Ext. cannab. ind. gr. ½
   Camphoræ gr. i.
   Confect. rosæ q.s.
   ft. pil. i.
   Occasionally as required. To relieve the pains of early stages.

Measles.

1. *Aconite*—in drop doses of the tincture every hour. Very useful at the onset.
2. ℞ Vin. ipecac. ʒi.
   Tr. camph. co. ʒiii.
   Liq. ammon. acet. ℥ss.
   Syrupi ℥ss.
   Aquam ad ℥ii.
   A teaspoonful every two hours. Useful in the catarrhal stage. (Children).
3. ℞ Liq. ammon. acetat. ʒss.
   Sp. ætheris nit. ♏x.-xx.
   Aq. camph. ℥ss.
   Every 4 or 6 hours. A mild diaphoretic in simple cases.
4. ℞ Potass. citrat. gr. v.-x.
   Vini ipecac. ♏v.-x.
   Tr. camph. co. ♏v.-x.
   Aquam ad ʒii.-iv.
   A simple cough mixture for early stages.
5. ℞ Potass. chlorat. gr. v.
   Acid. hydrochlor. dil. ♏iii.
   Syrupi limonis ʒss
   Aquam ad ℥ss.
   Every 3 or 4 hours. In measles of low type, with brown dry tongue and prostration. (Children).
6. *Bromide of potassium*—useful as a sedative, and to procure sleep in cases with active nervous symptoms and wakefulness.
7. *Tepid bath* or *tepid wet-sheet-pack* useful in high fever, and for early delirium. To be followed by general inunction.
8. *Mustard baths*—useful on sudden retrocession of the rash.

Melæna (*see Hæmorrhage*).

Meningitis.

1. *Active purge*—desirable at outset of the acute form, *e.g.* :—
   ℞ Hydrarg. subchlor. gr. ii.
   Ext. jalap. gr. viii.
   ft. pil. ii.
2. Head to be shaved, and ice applied, or leeches behind ears in acute cases.
3. ℞ Tr. physostigmatis (U.S. Ph.) ♏x.
   Ext. ergot. liquid. ♏xx.
   Aquam ad ℥ss.
   Every 3 or 4 hours. Useful in early stages of acute form. (Davis, *Chicago*).

4. ℞ Potass. bromidi gr. v.
Aq. et syrup. ʒii.
Every 3 or 4 hours. Useful in acute meningitis of children; chloral hydrat. gr. ii.-iii. may sometimes be added with advantage if much pain.

5. ℞ Ext. ergot. liquid. ♏xx.
Sp. ammon. aromat. ♏xx.
Aquam ad ℥ss.
Ever four hours, in early stages of acute form.

6. ℞ Potass. iodid. gr. ii.
Potass. bromid. gr. iv.
Tr. aurantii ♏xx.
Aquam ad ℥ss.
Thrice daily in later stages.

7. *Opium or morphia*—given internally or as a hypodermic injection. Useful in acute meningitis of adults; especially in epidemic form.

Mercurial Poisoning.

1. ℞ Potass. iodid. gr. v.-x.
Tr. belladon. ♏x.
Infus. calumbæ ℥i
Thrice daily.

2. ℞ Sulphur. precip. ℈ii.-iv.
Potass. chlorat. ℈ii.-ʒi.
Liq. morph. hyd. ʒi.-iss.
Mist. amygdalæ, ℥viii.
℥i. every three or four hours. To be well shaken. An excellent formula in ptyalism.

3. *Atropine*—the hypodermic injection of ♏i.-ii. of liq. atropinæ sulph.
Useful to check flow of saliva.

4. ℞ Potass. chlorat.
Boracis āā ʒiss.
Glycerini ℥iss.
Aquam ad ℥viii.
A useful mouth wash.

Myalgia.

1. Lin. belladon. or lin. opii useful external applications.

2. ℞ Ammon. chlorid. gr. x.-xx.
Aquæ ℥i.
Thrice daily. Highly recommended by Dr. Anstie.

3. ℞ Potass. nitratis gr. v.-x.
Pulv. ipecac. co. gr. iii.
To be taken as a powder every 4 hours.

Neuralgia.

1. ℞ Ung. veratrinæ.
℞ Ung. aconitinæ.
℞ Morphinæ gr. i.-ii.
Oleic acid ʒi.
℞ Lin. opii.
℞ Lin. aconiti.
℞ Lin. belladon.
Useful local applications in neuralgia.

2. *Hypodermic injections* of morphia or atropia, or of both combined.

3. ℞ Quin. sulph. gr. i.
Ext. hyoscyam. gr. i.
Camphoræ gr. i.
Confect. rosæ q.s.
ft. pil. i.
Every four hours.

4. ℞ Liq. arsenici hyd. ♏v.
Tr. ferri perchlor. ♏v.
Sp. chloroformi ♏x.
Aquam ad ℥i.
Thrice daily after meals.

5. ℞ Tr. gelsemii ♏xv.-xx.
Aquam ad ℥ss.
Every four hours in toothache.

6. ℞ Butyl-chloral hydr. gr. v.
Syrupi aurantii ʒss.
Aquam ad ℥ss.
Occasionally as required.

7. *Quinine*—in a single dose of gr. ii.-iv. during a paroxysm. In smaller doses combined with iron is a useful tonic in chronic cases.

8. *Phosphorus*—in doses of gr. $\frac{1}{100}$ to $\frac{1}{30}$ every three hours. Useful in many forms of neuralgia.

9. *Faradization* of painful spot.

Otorrhœa.

1. Careful *syringing*, followed by insufflation of boracic acid.

2. *Glycerine of tannin*—an excellent application in catarrh of external meatus.

3. ℞ Lotio plumbi acetat. dil.
℞ Lotio zinci sulph. (gr. ii. ad ℥i.).
℞ Lotio aluminis (gr. ii.-x. ad ℥i.).
Useful lotions for the ear passages.

4. A small *blister behind the ear* is often beneficial in obstinate cases.

5. *Tonics* and *cod-liver oil* often required.

Ozæna.

1. Frequent irrigation with tepid water or one of the following solutions:—
Weak Condy's fluid.
or Carbolic acid (1 or 2 per cent. solution).
or Alum (ʒi. ad aq. Oi.).
or Tannin (ʒss.-i. ad aq. Oi.).

2. *Insufflation* with one of the following powders.
℞ Hydrarg. oxidi rubri *vel*
Hydrarg. ammon. 1 part
Pulv. sacch. alb. 60 parts.
or ℞ Acidi tannici 1 part
Sacch. alb. 2 parts.
or ℞ Camph. (pulv.) 1 part
Sacchari alb. 15 parts.

3. *Tonics* and *cod-liver oil* are often required.

Paralysis.

1. *Liq. strychniæ* — in doses of ♏iii. internally, thrice daily, or of ♏i. hypodermically, once daily, the quantity being gradually increased—is more generally useful than any other drug.

2. ℞ Potass. iodid. gr. v.-x.
Ammon. carb. gr. ii.
Liq. hydrarg. perchlor. ʒi.
Decoct. cinchon. ℥i.
Thrice daily. Useful in syphilitic and some chronic forms of paralysis.

3. *Phosphorus*—in dose of gr. $\frac{1}{100}$ to $\frac{1}{30}$ in pill, twice or thrice daily; recommended in hysterical paralysis.
or ℞ Zinci phosphid. gr. $\frac{1}{3}$
Ext. nucis vom. gr. $\frac{1}{2}$
Ext. gentian. gr. ii.
ft. pil. i.
A useful pill in similar cases.

4. ℞ Potass. bromid. gr. x.-xx.
Tr. belladon. ♏x.
Aquam camph. ad ℥i.
Thrice daily. In paralysis accompanying acute congestion of nerve-centres.

5. *Galvanism* or *Faradism* of affected muscles useful in various forms of peripheral paralysis. That form of application should be selected to which the muscles best respond. No use where the irritability has quite disappeared.

6. *Counter-irritation* of the spine exceedingly useful in diphtheritic paralysis and other chronic forms.

Parasitic Skin Affections.

SCABIES.

1. Thorough washing at night with soap and hot water followed by inunction with ung. sulphuris.

2. ℞ Sulphur. sublim. ʒi.
Potass. carb. ʒss.
Adipis benzoat. ℥i.
A stronger ointment.

3. ℞ Sulphur. præcip. ʒss.-i.
Adipis benzoat. ℥i.
For irritable sensitive skin, as in infants.

4. ℞ Styracis præp. ℥i.
Adipis ʒii.
Very useful in scabies.

5. *Tonics* and *cod-liver oil* needed in bad pustular forms, or when there is much eczema; and *at first* a weak carbolic acid lotion or ointment is preferable in these cases.

PEDICULOSIS.

1. ℞ Ung. hydrarg. ammon.
For all forms of pediculi. The ointment should be diluted if much eczema or pustulation.

2. ℞ Hydrarg. perchlor. gr. i.-ii.
Glycerini ʒii.
Aquæ ʒvi.
ft. lotio.
Useful for pediculi capitis or pubis.
Not advisable if open sores exist, or if skin be broken.

3. ℞ Staphisagriæ (pulv.) ʒii.
Olei olivæ ʒii.
Adipis ʒvi.
ft. ung.
Best application for pediculi corporis.

4. Nits of pediculi capitis may be best removed by washing first with soap and hot water, and then with spirits of wine or paraffin oil, followed by the use of a fine tooth-comb.

VEGETABLE PARASITES.

1. Blistering with acetum cantharides in early stage of ringworm.

2. ℞ Hydrarg. ox. rub. gr. iii.
Hydrarg. ammon. gr. x.
Sulphuris sublim. gr. xx.
Ung. simplicis ℥i.
ft. ung.
℞ Acidi carbolici
Glycerini āā ℥i.
℞ Hydrarg. perchlor. gr. ii.
Glycerini ʒii.
Ung. cetacei ℥i.
ft. ung.
℞ Acid. carbolic. 1 part
Ung. hyd. nitrat. 1 part
Mix and then add
Ung. sulphuris 1-2 parts.
℞ Thymol ʒss.
Chloroformi ʒii.
Ol. olivæ ʒvi.
℞ Hydrarg. oleat. 5-10 per cent.
Useful parasiticide applications.

3. ℞ Sodii hyposulphitis ʒiv.
Glycerini ʒii.
Aquam ad ℥vi.
ft. lotio.
In tinea versicolor (chloasma), and other forms.
or Acid. sulphurosi (B.P.).

4. ℞ Ung. hydrarg. nit. ʒiv.
Creasoti ♏iv.
Glycerini ʒii.
Adipis ℥i.
ft. ung.
In tinea sycosis, and other forms.

5. ℞ Iodine ʒii.
Colourless oil of tar ℥i.
To be applied with a camel's hair brush in tinea tonsurans and circinata.

6. ℞ Tr. cantharid. ℥ii.
Acid. acet. fort. ℥i.
Glycerin. ʒiv.
Sp. rosmarin. ℥i.
Aq. rosæ ad ℥viii.
ft. lotio.
or ℞ Tr. cantharid. ℥i.
Tr. capsici ℥i.
Aq. rosæ ℥i.
ft. lotio.
Useful stimulant lotions in alopecia areata.

Perspirations, profuse.

1. *Liq. atropinæ sulph.*—in dose of ♏i.-ii. hypodermically injected is the promptest remedy.
or ℞ Ext. belladon. gr. ½
Ext. gentian. gr. ii.
To be taken at bed-time.

2. ℞ Acid. gallic. gr. ii.
Ext. cannab. ind. gr. ½
Confect. rosæ gr. i.
For a pill to be taken at bed-time.

3. ℞ Zinci oxid. gr. ii.
Ext. conii gr. iii.
For a pill to be taken at bed-time.

4. ℞ Quininæ sulph. gr. iii.
Zinci sulph. gr. i.
Acid. sulph. dil. ♏x.
Aq. et syr. aurant. ad ℥iss.
For a draught at bed-time.

5. ℞ Pulv. ipecac. co. gr. v.
As a powder, the last thing at night. If made as a pill, ext. belladon. gr. ¼ may be added with advantage.

Phthisis.

1. ℞ Ammon. chlorid. ʒi.
Syrupi scillæ ʒiii.
Infus. senegæ ad ℥vi.
℥ss. thrice daily. A good stimulating expectorant in acute phthisis.

2. ℞ Quininæ sulph. gr. i.
Pulv. digitalis gr. i.
Pulv. opii gr. ½
ft. pulv.
Every 4 or 6 hours, in acute phthisis. To be accompanied with the application of iced cloths to the abdomen. (Anderson).

3. ℞ Sodii hypophosphit. gr. vi.
Syrupi ʒii.
Aquam ad ℥i.
Once or twice daily.
℞ Calcii hypophosphit. gr. vi.
Syrupi ʒii.
Aquam ad ℥i.
Once or twice daily.
℞ Sodii hypophos.
Calcii hypophos. āā gr. viii.
Infusi cascarillæ ℥i.
Twice a day after meals.
Of no use in acute inflammatory, or very advanced phthisis. These medicines should be kept in a dark place.

4. *Cod-liver oil*—in doses of ʒi.-iv. thrice daily. Sir W. Foster recommends the addition of ether to the oil (ʒiiss. ad ℥iv.).

5. *Liq. arsenicalis* — in doses of ♏ii.-iv. every four hours well diluted; sometimes of great service in tubercular phthisis.

6. *Pancreatic emulsion* (Dobell). A table-spoonful from 1 to 2 hours after meals in milk.

7. ℞ Picis liquid. gr. ii.
Pulv. glycyrrhiz. co. q.s.
One pill three or four times daily for profuse fœtid expectoration.

8. ℞ Syrupi ferri iodid. ♏xx.-ʒss.
Thrice daily in water.

9. ℞ Morph. hyd. gr. ⅓
Acid. hydrocyan. dil. ♏v.
Acid. hydrochlor. dil. ♏v.
Oxymel scıllæ ℥ss.
Aquam ad ℥i.
A teaspoonful occasionally. A useful cough linctus.

10. ℞ Pulv. bismuthi nit.
Pulv. ipecac. co. āā gr. v.
As a powder 3 or 4 times daily as required. For diarrhœa.

11. ℞ Acid. sulph. dil. ♏xv.
Tr. opii ♏v.
Glycerini ℥i.
Decoct. hæmatoxyli ad ℥i.
Occasionally as required. For diarrhœa.

12. *Counter-irritation* — by iodine painted under clavicles; in chronic form to allay cough and to check secretion.
For night-sweats, see "*profuse perspiration.*"

13. For hæmoptysis, see "*hæmorrhage.*"

Pleurisy.

1. *Tr. aconite*—in drop doses every half to one hour in early acute stage.

2. *Applications* of hot fomentations or turpentine stupes in early acute stage.

3. ℞ Potass. acetat. gr. xv.
Sp. ætheris nitros. ʒss.
Vini ipecac. ♏v.
Syrupi tolut. ʒss.
Aquam ad ℥ss.
Every 6 hours. Useful in subacute pleurisy.

4. *Counter-irritation*—by application of iodine, croton-oil liniment, or flying blisters to affected side.
   To encourage absorption of effusion.
5. *Tonics*—especially the syrup of the iodide of iron and cod-liver oil.
   In chronic cases.

Pneumonia.

1. *Tr. aconite*—one or two drops every half hour for 2 hours, then less frequently.
   Quinine—in single doses of gr. v.-x. together with gr. $\frac{1}{4}$ to $\frac{1}{3}$ of morphia, followed every 3 or 4 hours by smaller doses without morphia.
   Recommended at first onset of acute pneumonia.
2. ℞ Liq. ammon. acet. ʒii.-iv.
   Vin. ipecac. *vel*
   Vin. antim. ♏x.-xv.
   Liq. morph. ♏x.-xv.
   Aquam ad ℥i.
   Every 4 or 6 hours.
   A useful formula during early acute stages, with much pain and restlessness.
3. ℞ Ammon. carb. gr. v.
   Sp. chloroform. ♏xx.
   Aq. camph. ad ℥i.
   Every 3 or 4 hours. In acute pneumonia of the aged and feeble.
4. Hot fomentations and turpentine stupes to relieve pain of side.
5. ℞ Ammon. carb. gr $\frac{1}{2}$-i.
   Vin. ipecac. ♏ii.
   Tr. aconiti ♏i.
   Aq. et syrup. ad ʒi.
   Every three or four hours. Useful in acute pneumonia of young children (early stages).
6. Hot mustard bath invaluable in many cases of acute pneumonia and bronchitis in children.
7. *Opium*, especially useful in the first indications of delirium with restlessness and tremor. Stimulants are also needed (adults).
8. *Quinine*, in doses of gr. iii.-v Every 3 or 4 hours. Especially in asthenic pneumonia of the aged.

Pruritus, Prurigo, and Itching.

1. ℞ Sodii carb. ʒi.
   Aquæ Oi.
   ft. lotio.
   ℞ Potass. cyanid. ʒi.
   Aquæ Oi.
   ft. lotio.
   ℞ Acid. hydrocyan. dil. ʒii.-iv.
   Glycerini ℥ii.
   Aquam ad Oi.
   ft. lotio.
   Useful sedative lotions when the skin is not broken.
2. ℞ Glycerini ℥ii.
   Solution (saturated) of boracic acid Oi.
   In various itching affections—varicose eczema, pruritus, pudendi, lichen, &c.
3. ℞ Acidi carbolici ʒi.
   Ung. zinci ox. ℥i.
   ft. ung.
   Useful in eczema (chronic) with much itching. To be diluted in sub-acute forms.
   ℞ Liq. carbonis detergens ʒss.-i.
   Glycerini ʒi.
   Aquam ad ℥x.
   Another formula for same purpose.
4. Solutio argent. nit. (gr. v. ad ℥i).
   To be applied frequently.
   Ung. hyd. subchlor.
   Lotio nigra.
   Useful applications in pruritus ani and pudendi.

Psoriasis.

1. *Arsenic*—Liq. arsenicalis in increasing doses thrice daily after meals.

2. ℞ Picis liq. ♏ii.
Pulv. aromat. q.s.
ft. pil. i.
Thrice daily. Especially where arsenic is ill-borne.

3. *Tonics* and *cod-liver oil*—especially in psoriasis of children.

4. ℞ Magnes. sulph. gr. xx.
Magnes. carb. gr. v.
Vini colchici ♏x.
Aquæ menth. pip. ℥i.
Thrice daily. In gouty psoriasis.

5. ℞ Ol. cadini *vel* picis liq.
Sp. vini rect.
Sapon. mollis æq. partes.
℞ Olei cadini
Olei morrhuæ æq. partes.
℞ Ung. picis (B.P.).
To be diluted if necessary.
℞ Acidi carbolici ʒi.
Ung. zinci ℥i.
ft. ung.
Useful local applications, when the skin is not too irritable or inflamed.

Purpura.

1. ℞ Quin. sulph. gr. i.
Tr. ferri perchlor. ♏x.
Liq. arsenic. hydrochlor. ♏iii.
Syrupi zingib. ʒi.
Aquam ad ℥i.
Twice or thrice daily.

2. ℞ Ext. ergot. liq. ♏xx.
Tr. ferri perchlor. ♏x.
Sp. chloroformi ♏x.
Aquam ad ℥i.
Thrice daily.

3. ℞ Ferri sulph. exsic. gr. ii.
Quin. sulph. gr. i.
Ext. nucis vom. gr. ½
Confect. rosæ q.s.
ft. pil. i.
Thrice daily.

4. ℞ Olei terebinth. ♏x.-xx.
Mist. amygdalæ ℥i.
Thrice daily.

Rheumatism, acute.

1. ℞ Salicin. gr. xx.
Aquæ ℥i.
Every three hours.

2. ℞ Sodii salicylat. gr. x.-xx.
Aquæ, ℥i.
Every three or four hours.

3. ℞ Sodii bicarb. ʒiss.
Potass. acetat. ʒss.
Liq. ammon. acetat. ʒiii.
Aquæ ℥iss.
Every 4, 6, or 8 hours in effervescence with acid. citric. ʒss. till the urine is alkaline; then less frequently.
℞ Sodii bicarb.
Potass. acetat. āā ʒss.
Decoct. cinchon. ℥iss.
Thrice daily as symptoms subside.
℞ Mist. quininæ ℥i.
Thrice daily during convalescence.

4. ℞ Ext. opii gr. ii.
Ext. belladon. gr. ½
ft. pil. i.
At bed-time if great restlessness and pain.

5. ℞ Potass. bicarb. gr. xx.
Potass. iodid. gr. v.
Decoct. cinchon. ℥i.
Thrice daily in chronic cases.

6. *Tonics*—during convalescence.
℞ Ammon. carb. gr. v.
Tr. lavand. co. ʒi.
Infusi cinchon. ℥i.
Thrice daily. An excellent formula.

Rheumatism, chronic.

1. ℞ Potass. iodid. gr. v.-x.
Ammon. carb. gr. ii.
Inf. calumb. ℥i.
Thrice daily. Especiallv where pains are worse at night.

2. ℞ Mist. guaiaci ℥i.
Thrice daily. Especially where pain is relieved by warmth.

3. ℞ Tr. acteæ racemos. ʒss.-i.
Aquam ad ℥i.
Three or four times daily.

4. ℞ Liq. sodii arseniat. ♏iii.
Vini colchici ♏x.
Tr. cinchon. co. ʒi.
Aquam ad ℥i.
Thrice daily after meals. In some forms.

5. ℞ Sodii sulphat.
Sulphuris præcip. āā ʒss.
Every morning in a tumblerful of milk and water.

6. ℞ Ammon. chlorid. gr x.-xx.
Infus. gentian. co. ℥i.
Thrice daily. Especially in muscular rheumatism.

7. *Turkish baths.*

8. *Cod-liver oil.*

Rickets.

1. *Cod-liver oil* should be given in small doses (♏xx.-ʒss.) at first.

2. Syr. ferri phosph. co. ʒss.-i.
or Syr. ferri iodid. ♏xx.-ʒss.
or Vini ferri ʒss.-i.
In water thrice daily.

3. ℞ Calcii phosph. gr. iii.
Bismuthi nitrat. gr. iii.
Sacch. albi ʒss.
Twice daily. Especially with tendency to diarrhœa.

Scarlet Fever. (*Children*).

1. *Tr. aconiti.*—One drop every hour or oftener at outset of fever.

2. ℞ Potass. chlorat. gr. iii.
Acid. hydroch. dil. ♏x.
Tr. aconiti ♏ii.
Aq. et. syrup. ad ʒi.
Every 3 or 4 hours in early stages.

3. ℞ Ammon. carb. gr. iii.
Aquæ ʒi.
Every 2 or 3 hours in a little milk.

4. *Tepid sponging* and *baths*, followed by inunction. In early delirium, and during desquamative stage.

5. *Quinine*—in doses of gr. ii.-iv. Thrice daily. In malignant cases with delirium and restlessness. If not retained, give treble dose by rectum.

6. ℞ Potass. chlorat. gr. v.
Tr. ferri perchlor. ♏v.
Glycerini ʒss.
Aquam ad ℥ss.
Every 3 or 4 hours. In scarlatina anginosa. Stimulants and good food also needed.

7. *For scarlatinal nephritis* :—
Warm baths.
Saline aperients.
℞ Potass. bitart. gr. xv.-xx.
Vini ipecac. ♏v.
Sp. æther. nit. ♏xv.
Infusi digitalis ʒss.-i.
Aquam ad ℥ss. (*Children above 5 years*).
Three times daily.

Scrofulosis.

1. *Cod liver oil.*

2. *Iron* — various preparations. Syrupi ferri phosph. — Syr. ferri iodid. - Vini ferri — or Parrish's chemical food recommended.

3. ℞ Calcii chlorid. (cryst.) gr. x.-xx.
Thrice daily in milk after meals.

4. ℞ Calcii phosph. gr. iii.
Thrice daily in milk.

5. ℞ Calcii sulphid. gr. $\frac{1}{10}$
Sacch. alb. q.s.
Every 2 or 3 hours.
In strumous suppurating sores.

Sleeplessness.

1. *Opium.*—Dose to be regulated according to age. Chiefly indicated in sleeplessness from acute disease. Where there is also much excitement the further addition of antimony desirable, thus :—

℞ Tr. opii ♏xv.
Vin. antimon. ♏xx.
Aquam camph. ad ℥i.
Every 4 or 6 hours.

℞ Pulv. opii gr. i.
Pulv. antimonial. gr. v.
ft. pulv.
To be taken at bed-time.

Opium should not be given for chronic sleeplessness unaccompanied by disease, nor in cases of albuminuria.

2. *Bromide of potassium.* Dose gr. xv.-xxx. or more. Useful in sleeplessness from worry, irritation, and excitement. Often advantageously combined with chloral.

3. *Chloral hydrate.* Dose gr. xv.-xxx. or gr. iii.-x. in children. Useful where opium is contra-indicated. Should be avoided or given with caution where the circulation or respiration is embarrassed.

4. *Alcohol* with light nourishment of great service in the sleeplessness of the aged and feeble. Also in certain low and malignant types of fever. To be taken towards bed-time.

5. *Hyoscyamus.* The tincture (♏v.-x.) is a useful sedative for young children. May be combined with pot. brom. and camphor water.

Small-pox.

1. ℞ Acid. salicylici gr. xx.
Sodii bicarb.
Ammon. carb. āā gr. iv.
Aquæ ℥i.
Every 3 or 4 hours. In early stages (adults). Later on add ferri et am. cit. gr. v. to each dose. A tepid bath each day ; after which body to be painted over with :—

℞ Acid. carbolic. ℥i.
Glycerini ℥i.
Gelatini ℥i.
Aquæ ℥xxvi.

As soon as pustules fully form, prick and apply gelatine lotion. During exfoliation a warm, slightly alkaline bath daily. (*Prideaux*).

2. *Saline aperients*—useful at the first onset.

3. ℞ Quin. sulph. gr. i.
Acid. sulph. dil. ♏xii.
Syrupi aurantii ʒss.
Aquam ad ℥i.
Every 4 hours where there is much debility and prostration. Also good food and alcohol.

4. *Opium* or *morphia* in moderate doses, combined with bromide of potash in the early delirium from small-pox in the intemperate.

4. *Warm baths*—of great service.

6. *Steel* and *quinine*—during convalescence.

Spinal irritation.

1. *Counter-irritation* to spine is frequently most valuable, especially in mild cases.

2. *Tonics* and *cod-liver oil.*

℞ Zinci phosphid. gr. $\frac{1}{2}$
Ext. nucis vom. gr. $\frac{1}{10}$
Ext. gentian. gr. ii.
ft. pil.
Three or four times daily.

℞ Zinci oxidi gr. ii.
Confect. rosæ q.s.
ft. pil.
Three or four times daily.

℞ Liq. arsenicalis ♏iii.
Tr. ferri perchlor. ♏x.
Aquam ad ℥i.
Thrice daily after meals.

3. *Hot-water bags* to spine.
4. *Galvanism* — applied to spinal region.

Stomach, ulcer of.

1. ℞ Bismuthi carb.
 Sodii bicarb. āā gr. v.
 Acid. hydrocyan. dil. ♏iii.
 Aq. et mucilag. ad ℥ss.

 Three or four times daily. To relieve pain and vomiting. Liq. morph. may be often added with benefit.
2. ℞ Liq. arsenicalis ♏i.-ii.

 Recommended by Ringer to check pain and vomiting. To be taken in water before food.
3. ℞ Argenti nitrat. gr. $\frac{1}{4}$
 Ext. opii gr. $\frac{1}{2}$
 Ext. hyoscyam. gr. iss.
 ft. pil.

 Thrice daily.
4. *Sulphate of Soda*—in aperient doses, or in form of Carlsbad water, to be taken early in morning to clear out stomach.
5. *Counter-irritation* to epigastrium in persistent vomiting.
6. *Peptonised* gruel and beef-tea. See *Brit. Med. Jour.*, 1880, vol. i., pp. 647, 683.
7. *Nutrient enemata, e.g.*
 Strong beef-tea 4-6 oz.
 Cream 1 oz.

 The addition of 1 teaspoonful of liq. pancreaticus (Benger) is desirable; and if the rectum be irritable, ♏v.-x. of laudanum.

Stomatitis.

1. ℞ Potass. chlorat. gr. x.
 Tr. ferri perch. ♏v.
 Acid. hydroch. dil. ♏v.
 Aquam ad ʒi.

 Three, four, or more times a day (ʒi.-ii.) for children, in all forms of stomatitis.
2. *Glycerine of borax*—a good local application in many forms.
3. ℞ Sodii sulphit. ʒi.
 Aquæ ℥i.

 A useful mouth-wash in thrush.
4. *Glycerine of tannin*—as a local application in ulcerative stomatitis.

Syphilis.

1. *Mercury*—in primary and secondary syphilis. The following are some of the chief forms of administration :—

 ℞ Hydrarg. cum cretâ gr. $\frac{1}{4}$-i.

 As a powder with sugar, or a pill with ext. gentian. three times a day.

 ℞ Pil. hydrarg. subchlor. co. gr. v.

 Twice or thrice daily.

 ℞ Ung. hydrargyri ʒi.

 To be used as an inunction daily.

 ℞ Hydrarg. subchlor. gr. xv.-xxx.

 Volatilised by heat, and used with vapour bath

 N.B. As soon as the gums are in the least affected, smaller doses should be taken. Slight mercurial advisable for at least two years after primary sore, in order to eradicate disease.
2. ℞ Liq. hydrarg. perch. ʒi.
 Decocti sarsæ co. *vel*
 Decocti cinchon. ℥i.

 Thrice daily in chronic cases.
3. ℞ Potass. iodid. gr. v.
 Ammon. carb. gr. ii.
 Liq. hydrarg. perch. ʒi.
 Inf. gentian. ℥i.

 Thrice daily. A useful formula.
4. ℞ Hydrarg. perchlor. gr. i.
 Glycerini boracis ℥i.

 To be painted on obstinate secondary ulcers of mouth. Added to half a pint of water may be used as a gargle.
5. ℞ Liq. Donovan ♏x.-xv.
 Aquam ad ℥i.

 Thrice daily. Especially in scaly skin affections.

6. *Iodide of potassium* gr. v.-x. Thrice daily combined with bark or other vegetable bitters. Especially in tertiary gummata.

7. ℞ Syr. ferri iodid.
Ext. sarsæ liquid. āā ʒss.
Thrice daily in water.

8. General *tonics* and *cod liver oil*, preferable at first to specific treatment in long-standing cachectic cases.

Tape-worms.

1. ℞ Ext. filicis liq. ♏xxx.-ʒi.
Syrupi zingiber. ʒii.
Mucilag. tragac. ℥i.
Aquam ad ℥ii.
As a draught at early morning.

Throat Affections.

1. *Aconite*—drop doses of tincture every ½ or 1 hour in early stages of relaxed throat or tonsillitis.

2. ℞ Potass. chlorat. gr. x.
Tr. ferri perchlor. ♏x.
Sp. chlorof. ♏x.
Aquam ad ℥i.
Every four hours. In sore throat.

3. Useful gargles in inflammatory conditions of throat:—
℞ Aluminis exsic. gr. 80
Tr. myrrh. ʒi.
Aquam ad ℥viii.
℞ Aluminis exsic. ʒi.
Tr. capsici ʒi.
Aquam ad ℥viii.
In relaxed throat and tonsillitis.
℞ Creasoti ♏xx.
Mucilag. tragac. ℥iii.
Aquam ad ℥viii.
(Shake well).
In sloughing, foul ulcers of throat.

4. *Glycerine of tannin.* As an application to throat, very generally useful.

5. *Extract of belladonna*—well smeared over outside of throat, and a hot fomentation or poultice applied. In acute tonsillitis, to relieve pain.

6. *Quinine* and other *tonics* required on cessation of acute symptoms.

Typhoid Fever. (*See Fever*).

1. *Bath* of 95° cooled to 75° and continued for 20 minutes. To be repeated as often as necessary. For high fever. Most useful in early stages.

2. *Sponging the body* with tepid water, ice being applied to head.
In extreme restlessness, delirium, and wakefulness.

3. ℞ Quinine gr. v.-viii.
Every half-hour till temperature falls or distinct cinchonism produced. In high fever.

4. ℞ Bismuth. nitrat. gr. v.
Pulv. ipecac. co. gr. v.
Every 4 or 6 hours as required. In excessive diarrhœa.

5. *Enema opii*—in excessive diarrhœa.

6. ℞ Pulv. opii gr. i.
Cupri sulph. gr. ¼
Conf. rosæ q.s.
ft. pil.
As required in excessive diarrhœa.

7. ℞ Tr. opii
Olei terebinth.
Tr. chloroformi co. āā ♏x.-xv.
Acidi tannici gr. x.
Mucilag. et aq. ℥i.
Every 4 hours as required. In hæmorrhage.

8. ℞ Ol. terebinth. ♏x.
Mist. amygdalæ ad ℥i.
Every 3 or 4 hours. An excellent formula for routine use in all cases; but especially

towards end of second week, with dry, brown, cracked tongue, and tympanitis. Also in protracted diarrhœa. Also in hæmorrhage.

9. *Strong black coffee*—2 or 3 table-spoonfuls occasionally. As a stimulant in place of alcohol.

10. ℞ Potass. chlorat. ʒi.
Glycerini ℥ii.
Aquam ad ℥iv.
A pleasant mouth-wash.

11. Chloral hydrate, bromide of potash, morphia, and camphor. Useful sedatives for restlessness.

Typhus Fever. (*See Fever*).

1. ℞ Liq. opii sed. ♏v.
Antim. tart. gr. $\frac{1}{12}$
Aq. camph. ℥ss.
Every hour till sleep is induced. When delirium is active and prolonged.
℞ Liq. opii sed. ♏v.
Sp. æth. sulph. co. ♏xx.
Aq. camph. ℥i.
Every hour till sleep is induced. When delirium is wandering and pulse feeble.
N.B. Where dyspnœa, lividity, contracted pupil, and cerebral congestion, opium is wholly contra-indicated.

2. *Quinine.* To reduce temperature (*see Typhoid*).

3. *Cold baths.* To reduce temperature (*see Typhoid*).

4. ℞ Tr. digitalis
Sp. æth. sulph. co.
Tr. belladon. āā ♏x.
Aquæ ℥i.
Every 4 hours. In cardiac feebleness.

Urine, Incontinence.

1. Attend to thread-worms, vesical calculus, adherent or long prepuce—if present. Especially in children.

2. *Belladonna*—10 to 15 drops of the tinct. thrice daily, with camphor water. In simple incontinence of children.

3. ℞ Potass. cit. gr. x.
Liq. potass. ♏v.
Tr. hyoscyam. ♏v.
Aquam camph. ad ʒii.
Thrice daily in water. For incontinence depending on lithiasis. (Children).

4. *Tr. cantharides.* One or two drops in certain cases among the aged. 3 or 4 times daily (Ringer).

5. *Tonics.* Especially iron or strychnia.

Urticaria.

1. Aperient, and perhaps emetics in early stage of acute cases.

2. ℞ Magnes. sulphat. ʒss.
Sodii bicarb. ʒss.
Tr. zingiberis ♏x.
Aquæ ℥i.
Thrice daily with acid. tartaric. gr. xx.
℞ Ferri sulph. gr. i.
Magnes. sulph. gr. xx.
Acid. sulph. dil. ♏v.
Tr. zingiber. ♏xv.
Aquam menth. pip. ad ℥i.
Thrice daily.
Useful formulæ in acute or subacute cases.

3. ℞ Bismuthi nitrat. gr. v.
Sodii bicarb. gr. xx.
Pulv. zingib. gr. ii.
Thrice daily (in water if preferred).

4. *Arsenic.* Useful in some chronic cases. Iron often a good addition.

5. ℞ Acid. carbolic. ʒi.
Ung. zinci ℥i.
ft. ung.
℞ Tr. benzoin. co.
To be painted on the skin.
Useful applications to relieve itching.

Vomiting.

1. ℞ Sodii bicarb. gr. iii.
Tr. nucis vom. ♏½
Sp. chloroform. ♏i.
Aq. et syrup. ad ʒi.
Three or four times daily. Useful in chronic vomiting in children with gastric catarrh.

2. ℞ Tr. nucis vom. ♏i.
Every ½ or 1 hour as required.
In chronic vomiting of nervous women with indigestion.

3. ℞ Bismuth. carb. gr. v.-x.
Sodii bicarb. gr. x.
Acid. hydrocyan. dil. ♏iii.
Mist. amygdalæ ℥i.
Thrice daily in some forms of gastric vomiting. Liq. morph. may be added if much pain, as in cancer of stomach; or sp. ammon. amorat. if too "cold."

4. ℞ Creasoti ♏i.-ii.
Pulv. aromat. gr. ii.
Mucilaginis q.s.
ft. pil.
Twice or thrice daily.

5. ℞ Liq. arsenicalis ♏i.
In water before food, in vomiting of drunkards (Ringer).

6. ℞ Vin. ipecac. ♏i.
Every ½ or 1 hour in water as required. In almost all cases of reflex vomiting.

Whooping Cough.

1. *Salines* and *expectorants* in catarrhal stage.

2. ℞ Acidi carbolic. ♏i.
Ess. menth. pip. ♏½
Syrupi ʒss.
Mucilaginis ʒss.
Every four or six hours. A very useful formula; may be combined as required with tr. hyoscyam. ♏v., or vin. ipecac. ♏ii.-iv., or acid. hydrocyan. dil. ♏½-i.
For children from 5 years and upwards.

3. ℞ Potass. bromid. gr. i.-v.
Tr. belladon. ♏v.-x.
Syrupi papaveris ♏xv.
Aquam ad ʒii.
Every 4 or 6 hours. For a child of four years and upwards. Children bear belladonna very well.

4. ℞ Quin. sulph. gr. i.-ii.
Acid. sulph. dil. q.s.
Tr. aurantii ♏x.
Syrupum ad ʒi.
Every three hours (Reynolds).

5. ℞ Ext. belladon. gr. i.
Aluminis ʒss.
Syrupi zingib. ʒi.
Mist. acaciæ ad ℥iii.
A teaspoonful thrice daily.

6. *Counter-irritation* to chest. A valuable adjuvant to any form of treatment, if much bronchial secretion. Turpentine liniment is a convenient preparation.

7. *Tonics* and *cod-liver oil*. In all chronic cases.

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